THE
AMERICAN
SURGEON

Subscription in the United States, $8.00

Vol. XVII, No. 10 October, 1951

THE EVALUATION OF PROTEIN-BOUND IODINE DETERMINATION IN THYROID DISEASE

T. C. Davison, M.D.*
A. H. Letton, M.D.
Atlanta, Ga.

Major deviations of thyroid activity from the normal can usually be easily diagnosed without the aid of the laboratory; however, the minor deviations many times may prove quite confusing and the laboratory is then called upon to help out, a call which too often the usual methods are not sensitive enough to answer. At times we have also been disappointed in the usual laboratory procedures; that is, the basal metabolic rate and cholesterol in the estimation of major deviations of thyroid activity. It seems that this has too often been true when one of our friends has referred a particular patient to us and we have wanted very badly for that test to bear out our clinical impression. With these deficiencies in mind we have continued our search for some laboratory procedure which would give us more accuracy and dependability.

Following the work of Salter and Bassett[1] and also Swenson and Curtis[2] we decided to see if protein-bound iodine determination could answer our problems. For years this test was far too complicated for the ordinary clinical laboratory, but at the meeting of *The American Goiter Association* in July, 1949, Connor, Curtis, and Swenson[3] introduced a simplified method of making this determination and upon our return home from that meeting, we busied ourselves in setting up the special apparatus necessary and in train-

*President, American Goiter Association.

Presented during the Hollywood Assembly of The Southeastern Surgical Congress, Hollywood, Fla., Apr. 11-14, 1951.

ing our technicians in this technic. This experience is the basis of our report today.

In general, the protein-bound iodine found in the blood stream of the patient who has not been taking large quantities of iodine medications represents the active principles produced by the thyroid gland. Therefore, any determination of the protein-bound iodine is a direct measure of the activity of the gland, and may be taken as an index of the level of circulating thyroid hormone. The basal metabolism rate, as you recall, is an indirect measure of the activity since it is a measure of the amount of oxygen used in metabolism, which is for the most part governed, in the basal state, by the activity of the thyroid. Thus, you can see how protein-bound iodine determination eliminates many of the variables in this problem.

Early reports, based on inadequate, tricky, involved analytical methods, led to considerable mistrust of the value of protein-bound iodine as a diagnostic aid. With the simplified type of analysis, which we will explain, the rechecks are quite accurate with a variation of about 5 per cent.[4]

There are many different procedures for determining protein-bound iodine, such as permanganate acid ash, acetone fractination, chromium trioxide oxidation and others. The method we use at present, which is a slight modification of the acid digestion-distillation method of Curtis,[4] is not only simpler, but also a little more accurate than the other methods.[5]

In this method 3 cc. of blood serum is precipitated with Somogyi reagent and centrifuged; then the supernatant fluid, which contains the inorganic iodides, is decanted. After this the precipitate is washed, then oxidized with chromic and sulfuric acid. Following this oxidation, phosphorus acid is added, liberating the iodine which is distilled over and caught in a potassium hydroxide trap. Ceric sulfate and arsenious acid are added, forming a canary yellow solution that is decolorized in the presence of iodine. The rate of decolorization depends on the amount of iodine present, the iodine acting as a catalyst. By using known concentrations of iodine the amount of iodine in the original sample can be calculated. The total procedure requires about three hours but does not require constant attention. This is actually less time consuming than some of the other common procedures; for example, the six hour glucose tolerance test. Apparently each laboratory must, after sufficient experience, set up their own mean normal deviations because of little difference in physical equipment and in standard solutions. It must be remembered that here we are dealing with a very minute amount of iodine; in fact, our average normal is 4.6 micrograms

per cent or 46/10,000 of one milligram of iodine in 100 cc. of blood serum. Since we start out with only 3 cc. of serum, then actually in this procedure we are handling only 3/100 of 46/10,000 or 138/1,-000,000 of one milligram of iodine. We have found, by compiling our statistics of the last two years, that our normal mean deviation is 3.2 to 5.6 micrograms per cent. This means that below 3.2 micrograms per cent we feel that there is too little circulating thyroid hormone and likewise over 5.6 micrograms per cent is too much. Thus it follows that values of less than 3.2 micrograms per cent denotes a state of hypothyroidism, while values over 5.6 micrograms per cent denotes hyperthyroidism. The extremes encountered in our experience were a low of 1.39 micrograms per cent and a high of over 12.7 micrograms per cent.

In an attempt to determine the accuracy of the protein-bound iodine determinations, we first eliminated the results of all patients who were not followed or on whom the diagnosis was not definitely proven either by pathological examination of the tissue or by the patient's response to therapy. We then listed all these results in one of three groups according to the proven diagnosis; that is, hypothyroid, euthyroid, or hyperthyroid. Inspection was then made to see how accurately the protein-bound iodine determination agreed with the final diagnosis. Fourteen determinations did not agree. On referring to the histories of these individuals it was found that 4 had been on propyl thiouracil prior to running the test and had very low protein-bound iodine values averaging 1.82 micrograms per cent. Five other patients had been taking iodine. Their protein-bound iodine values were increased and averaged 9.03 micrograms per cent. Three patients had previously undergone gallbladder series which caused their protein-bound iodine to be quite high. They averaged 11.4 micrograms per cent. The remaining 2 were apparently technical errors. In one case which presented a high protein-bound iodine, a subsequent reading showed a protein-bound iodine of proper level. In the other, a student nurse with Graves disease, the protein-bound iodine was 2.76 micrograms per cent. She was placed on Tapazole (1 methyl, 2 mercaptoimidazole-Lilly) the same day the first test was obtained and subsequent determinations would by virtue of this be modified. Thus, the error in which protein-bound iodine determinations did not agree with the proven diagnosis was 3.3 per cent.

In this same manner and on the same series of patients we also calculated our error in basal metabolic rate and cholesterol; that is, the number of times the basal metabolic rate and cholesterol did not agree with our diagnosis. The basal metabolic rate error was 22.2 per cent, which we realize is a little high; however, when you con-

sider that these tests are done in the laboratory located in our office, and that patients must come from their home which may be as many as 100 miles away that day, lie down and attempt to become basal, we feel it is not too far afield. The error of cholesterol was 29.1 per cent, which is easily understandable when you stop to enumerate the factors which control cholesterol level in the blood.

We have mentioned several factors which seem to cause discrepancy between the protein-bound iodine levels and the thyroid activity. Let us elaborate on this a little further. Pregnancy increases the protein-bound iodine.[6] This may theoretically be due to the addition of the fetal thyroid activity, which is well known to be considerably increased, as well as a probable increase in the action of the maternal metabolism. The administration of iodine also causes increased protein-bound iodine. This is shown in several instances where the patients had allergic manifestations and were given large doses of iodine.[7] It has been theorized[2] that inorganic iodine in large doses, probably in conjunction with certain enzyme systems within the blood, causes iodinization of other protein molecules without entering the metabolism of the thyroid gland, thus producing protein-bound iodine which does not have any direct effect on metabolism of the body. Intravenous pyelograms and gallbladder series utilize iodinated compounds and thus effect the protein-bound iodine level. The latter effects an increase in the protein-bound iodine for some months[6] because it is continuously spilled into the intestinal tract and then rapidly reabsorbed. The instillation of lipiodol[6] in the body has even longer lasting effect and may change the protein-bound iodine for several years.

It is an interesting sidelight to notice how quickly the antithyroid drugs reduce the protein-bound iodine, whereas it is remembered that it takes several weeks for a change to come about in the metabolic rate of the individual. This re-emphasizes the fact that the thyroid hormone acts peripherally on the tissues themselves, and probably has been built by the "Thyrenzymes"[8,9] into a different and larger protein molecule which is probably not reabsorbed back into the blood stream.

Conclusions

1. At present we favor the protein-bound iodine determination in estimating the activity of the thyroid gland because of the low rate (3.3 per cent) of error against 22.2 per cent error in the basal metabolic rate. It is particularly good in an office practice for the patient need not be in a basal state when the blood is drawn.

2. Protein-bound iodine cannot be relied on blindly to establish a

diagnosis but when used in the light of other evidence is quite helpful.

3. Protein-bound iodine is altered by rapid response to antithyroid drugs, as well as by iodine medication, intravenous pyelogram, gallbladder series or lipiodol injection.

REFERENCES

1. Salter, W. T., and Bassett, A. M.: Physiological interpretation of blood iodine fractions in terms of thyroid function in one hundred cases, Tr. A. Am. Physicians *56:*77, 1941.
2. Swenson, R. E., and Curtis, G. M.: Blood iodine in hyperthyroidism, Tr. Am. Goiter Assoc., 1948.
3. Conner, A. C.; Curtis, G. M., and Swenson, R. E.: Simplified method for determination of protein-bound iodine and its application, Tr. Am. Goiter Assoc., 1949, p. 435.
4. Conner, A. C., and others: Determination of blood iodine; a useful method for clinical laboratory, Surgery *25:*510 (April) 1949.
5. Barker, S. B., and Humphrey, M. J.: Clinical determination of protein-bound iodine in plasma, J. Clin. Endocrinol. *10:*1136 (Sept.) 1950.
6. Riggs, D. S.: Serum protein-bound iodine as diagnostic ridge, Tr. Am. A. Study Goiter, 1947, p. 137.
7. Danouski, T. S., and Greenman, J. H.: Alterations in serum iodine fractions during administration of potassium iodide, Tr. Am. Goiter Assoc., 1949, p. 154.
8. Salter, W. T., and Johnston, M. W.: Tracing thyroid hormone in peripheral tissue, Tr. Am. Goiter Assoc., 1948, p. 46.
9. Bassett, A. M.; Coons, A. H., and Salter, W. T.: Protein-bound iodine in blood. V. Naturally occurring iodine fractions and their clinical behavior, Am. J. M. Sc. *202:*516 (Oct.) 1941.

SURGICAL CORRECTION OF CONGENITAL EVENTRATION OF THE DIAPHRAGM

JOHN G. SHELLITO, M.D.
WAYNE C. BARTLETT, M.D.
Wichita, Kan.

THE surgical correction of eventration of the diaphragm in the infant has rarely been reported. Bisgard[1] first reported a surgical correction in an infant, 6 weeks old, in 1947. State[2] in 1949 reported a second case. The third report to appear in the literature was that of Butsch and Leahy[3] in 1950. According to the above authors, the first 2 patients suffered from a defect of the right diaphragm. Butsch and Leahy report the defect on the left side. The patient here presented suffered from a left congenital diaphragmatic eventration.

CASE REPORT

W. H., a male child, 5 months of age, was brought to the clinic by its mother. She stated that the child had not developed as well as it should; that he would become cyanotic upon exertion or crying and that he had intermittent difficulty in swallowing. The mother further stated that the child was a "poor nurser" and had frequent choking spells. The weight of the child at birth was 6 pounds 3 ounces, and at the time of surgery was 9 pounds 1 ounce.

Upon physical examination a few breath sounds were heard high in the left chest posteriorly. It was also possible to hear bowel sounds quite clearly in the remainder of the chest. The abdomen was small and scaphoid. The child had 12 Gm. of hemoglobin and 4,050,000 red blood cells. Urine examination was negative. X-ray examination of the chest (fig. 1a) revealed intestines, and what was thought to be a portion of the liver, in the left chest. It was questionable whether there was a diaphragm between the abdominal contents and the small portion of the left lung which could be discerned above them.

Following several small blood transfusions, the child was operated upon Jan. 18, 1950. A posterolateral incision was made over the ninth rib, which was resected subperiosteally, and the pleura opened through its bed. Upon entering the chest, it was found that the defect was due to a large eventration of the diaphragm which filled approximately two thirds of the left thorax (fig. 1b). The left lung, with the exception of the left upper lobe, was collapsed. An incision was then made in the abdomen as an extension of the chest incision; however, the costochondral cartilages were preserved anteriorly in order that the chest might be more rigid postoperatively. The contents of the sac, the left lobe of the liver, the stomach and portions of the small bowel and colon were then delivered into the abdominal cavity from below. It then became possible to dissect the diaphragm itself from the mediastinum, the pericardium and the medial aspect of the left pleural cavity to which it was firmly attached by adhesions. A small segment of the muscular portion of the diaphragm was found attached to the anteromedial portion of the left chest cage. This was preserved and approximately one half of the eventration itself was

From the Department of Surgery, the Wichita Clinic.

resected from the mid portion of the diaphragm. The remaining two flaps were used to create a new diaphragm for the child by imbrication (fig. 2a). Interrupted black silk sutures were used throughout. The chest was closed in

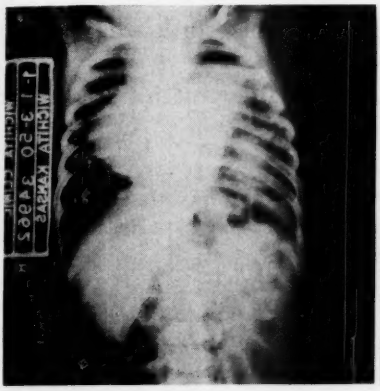

Fig. 1a. X-ray showing intestines and what was thought to be a portion of the liver in left chest.

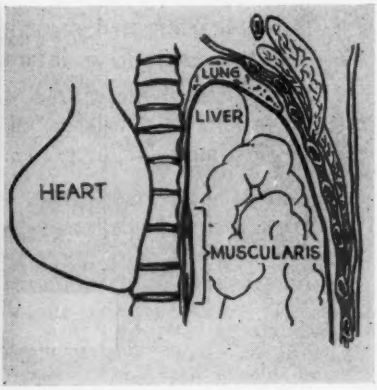

Fig. 1b. Drawing showing defect in left diaphragm.

the usual manner with two No. 12 urethral catheters connected to suction in order to create a negative intrathoracic pressure.

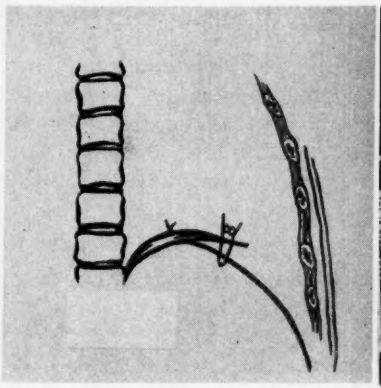

Fig. 2a. Drawing showing new diaphragm formed by imbricating flaps.

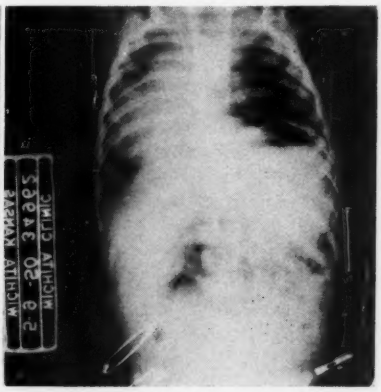

Fig. 2b. X-ray showing expanded left lung after surgery and heart in right chest.

Due to the fact that the lung did not completely expand at the time of surgery, bronchoscopy postoperatively was necessary on four occasions. The lung gradually expanded and filled the left chest cage (fig. 2b). The postoperative course was otherwise uneventful. The heart, which was in the right chest at the time of surgery, has remained in the same position. During the past year the child has had no noted cyanosis, dyspnea or dysphagia. He has continued to gain weight and at the present writing weighs 16 pounds.

Conclusions

1. A successful thoracico-abdominal approach for the repair of congenital diaphragmatic eventration is described.

2. To the best of our knowledge, this is the fourth successful repair of this anomaly in an infant.

3. It is felt that the thoracico-abdominal approach is necessary in those infants in which the diaphragm is attached to the chest cage, mediastinum and/or pericardium.

References

1. Bisgard, J. D.: Congenital eventration of diaphragm, J. Thoracic Surg. *16:*484 (Oct.) 1947.
2. State, D.: Surgical correction of congenital eventration of diaphragm in infancy, Surgery *25:*461 (March) 1949.
3. Butsch, W. L., and Leahy, L. J.: Technic for surgical treatment of congenital eventration of diaphragm in infancy, J. Thoracic Surg. *20:*968 (Dec.) 1950.

SUPRADUODENAL AND TRANSDUODENAL EXPLORATION OF THE COMMON BILE DUCT

MELL B. WELBORN, M.D.*
Evansville, Ind.

DURING the course of every cholecystectomy the surgeon must decide in his own mind as to whether or not the common bile duct should be opened and explored for stones. Some surgeons nearly always open the common bile duct and explore it, but the majority only explore it after due consideration for certain indications which they feel necessitate exploration. These indications are as follows: (1) the palpation of a stone in the common or hepatic ducts, (2) a dilated or thickened common duct, (3) jaundice, or a history of recent jaundice, (4) biliary colic, without stones in the gallbladder, and (5) small stones and the presence of a dilated cystic duct.

There is no doubt that opening and exploration of the common duct increases the difficulty and the hazards of biliary tract operative procedures. It is also true that some stones will be overlooked even with the duct opened and even after careful exploration. In a series of 439 choledochotomies, Buxton and Burk,[3] and others,[5] reported overlooked stones in 5.9 per cent of the patients. Most of these overlooked stones are in the pancreatic and ampullary portion of the common bile duct. This is the most difficult part of the duct system to explore adequately, and at times it is quite difficult to know whether or not the ampulla of Vater is patent. Under these circumstances, and occasionally in other conditions, combined supraduodenal and transduodenal exploration of the common duct is quite helpful.

Cattell[2] was one of the early advocates of combined supraduodenal and transduodenal exploration of the common duct in the presence of suspected impacted stones in the ampullary region, or of stricture about the papilla, and in some other cases. He devised a T tube with a long limb which could be extended through the ampulla into the duodenum. Mahorner[1] in 1949 again called attention to this method and pointed out that Cattell's procedure had not gained wide popularity, and apparently if the literature was to be used as a judge of whether or not the method had been widely adopted, it had not been done so. The method perhaps finds its greatest field of usefulness when the surgeon is exploring the common duct through a supraduodenal incision, and when he cannot be

*From the Surgical Service of the Welborn Memorial Baptist Hospital and The Welborn Clinic.

certain as to whether or not he has definitely established the patency of the papilla. This is sometimes very difficult to ascertain. One can palpate very carefully and still be in doubt as to whether or not the probe has passed into the duodenum. It is not uncommon also for false passages to be made into the duodenum. When there remains reasonable doubt as to whether or not the probe has passed into the duodenum, transduodenal exploration should be added to the operative procedure in order to visualize definitely the papilla and establish its patency. There is a tendency at times to hope that the duct system has been cleared of stones, and to feel that if it has not been, any remaining stones can be fragmented by ether instillations into the T tube. It is not wise to depend on this procedure, even though at times it may be found helpful under certain circumstances. Anastomosis between the biliary and intestinal tract as a substitute for removal of stones in the ampullary region should be avoided. A high percentage of such patients will have postoperative cholangitis with chills and fever.

Apparently this operative procedure has not been carried out more commonly due to the fear on the part of the surgeon of peritonitis, or the establishment of a duodenal fistula.[4] Surgeons have repeatedly warned against the dangers of opening the duodenum. Apparently, however, this danger has been exaggerated. Another objection to such exploration, particularly with division of the sphincter at the ampulla and the passage of a long-armed T tube into the duodenum, has been that of reflux of duodenal contents into the biliary tract. This, too, must be an uncommon finding, or if it does occur, it does not result in any serious symptoms.

Indications

There are several indications for combined transduodenal and supraduodenal exploration of the common duct. The most common indication is that in which choledochotomy is being carried out, stones have been found and there is doubt in the surgeon's mind as to whether or not the ampullary and pancreatic portions of the duct have been completely cleared of stones. The second indication is when the surgeon suspects that there is a stricture about the ampulla of Vater. The third indication is that which arises during resection of a duodenal diverticulum, and in which the relationship of the papilla to the diverticulum must be definitely established. The fourth indication is somewhat similar to the third, and is that which arises during resection for duodenal ulcer, and in which the surgeon fears that the patency of the papilla may be compromised by inversion of the duodenal stump. The fifth indication is that for tumors

of the ampulla of Vater, the adjacent area of the duodenum, or the head of the pancreas.

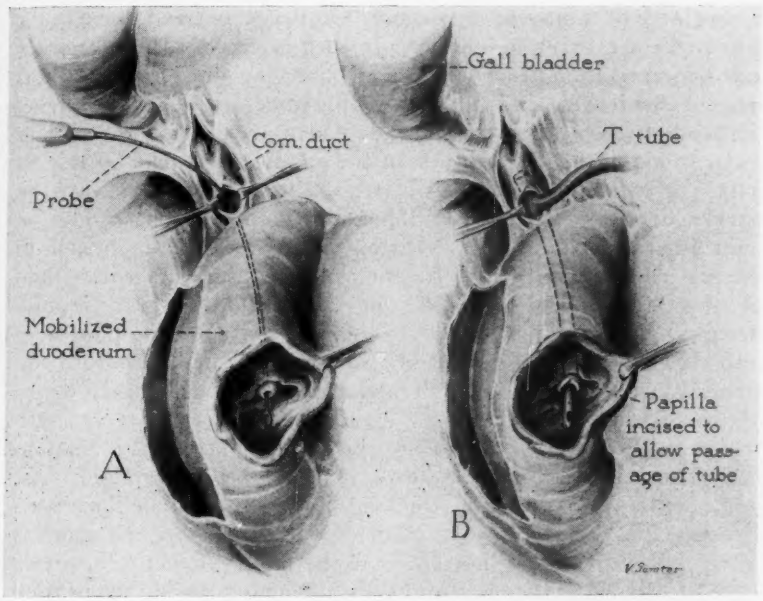

Fig. 1a. It is essential to open the peritoneum over the right side of the duodenum and to mobilize this structure quite well. Figure 1a demonstrates this maneuver, and shows the supraduodenal opening in the common duct, the longitudinal incision in the duodenum, and a probe traversing the distal end of the common bile duct.

Fig. 1b shows the T tube in place. The duodenal closure is carried out in a transverse manner. It must be meticulous.

Technical Considerations

There are certain maneuvers connected with the technic of this procedure which deserve comment. The artist has depicted the most essential ones in figures 1a and 1b. The most important consideration is wide mobilization of the duodenum by dividing the lateral peritoneal reflexion and then rotating the duodenum and the head of the pancreas medially towards the midline. This maneuver alone may help greatly in passing the probe from the supraduodenal incision in the duct into the duodenum. It must always be carried out before the duodenum is opened. The incision into the duodenum is made in a horizontal manner low down in the second portion over the region of the ampulla of Vater. The latter may be visualized by placing small Richardson retractors into the incision of the duo-

denum and grasping the region of the papilla with Babcock forceps and elevating it into the wound. If there is a tumor in this area it can be easily seen. A stone may be noted partially eroding its way through the duct into the duodenum. On the other hand, the papilla may be very difficult to identify. In such instances identification is aided by irrigating from above and observing the escape of fluid into the duodenum. In the presence of a stone, with partial obstruction and stricture formation, usually enough fluid leaks through in order to aid in the identification of the papilla. One may be able to pass a probe or catheter easily from above downward into the opened duodenum. If this is impossible, a small probe should be inserted at the papilla and passed superiorly towards the incision in the superior portion of the common duct. A small tube can then be tied to the end of the probe and the tube drawn into the duodenum. This tube may grasp any remaining stones and pull them into view, or it may be necessary to incise the papilla before the stones can be liberated into the lumen of the duodenum. In figure 1b, the artist has illustrated the division of the papilla in order to free a small stone in one case in which partial stricture formation had taken place. After the duct system has been thoroughly irrigated with a saline solution, the long limb of the T tube illustrated in figure 2 is drawn into the duodenum and the short end remains in that portion of the duct towards the liver. A meticulous, two layer closure of the duodenum is then carried out closing the incision in a transverse manner in order to prevent stricture formation. It is perhaps easiest to close the duodenal mucosa with a continuous stitch of 0000 catgut as a separate layer. The seromuscular coats are then closed with similar material and the suture line reinforced with a few very fine silk or cotton sutures. Following this, the supraduodenal portion of the duct is closed about the vertical end of the T tube. Cholecystectomy, if the gallbladder remains, may then be carried out.

Postoperative Care

The postoperative care in these patients is similar to that of the patients who have had ordinary choledochostomy. Since one limb of the T tube extends into the duodenum most of the bile usually escapes into the intestinal tract. The tube can be utilized for infusions of fluids such as saline and glucose into the duodenum. It should be irrigated twice daily with normal salt solution and can be clamped off when the patient is allowed to be up and about. Some bile-stained, rather thick appearing fluid resembling intestinal contents may reflux about the tube or through the lumen of the tube. In addition to irrigating the tube with saline solution a cholagogue

is advisable. These measures prevent the tube from becoming encrusted with bile salts and may prevent biliary stasis about the proximal end of the tube with the formation of biliary mud or even a small stone. Postoperative cholangiograms are usually of no value since the radio-opaque material so rapidly escapes into the duodenum that it is impossible to fill the proximal portion of the biliary tree. The length of time that the tube is allowed to remain in the duct varies somewhat, depending on the pathological condi-

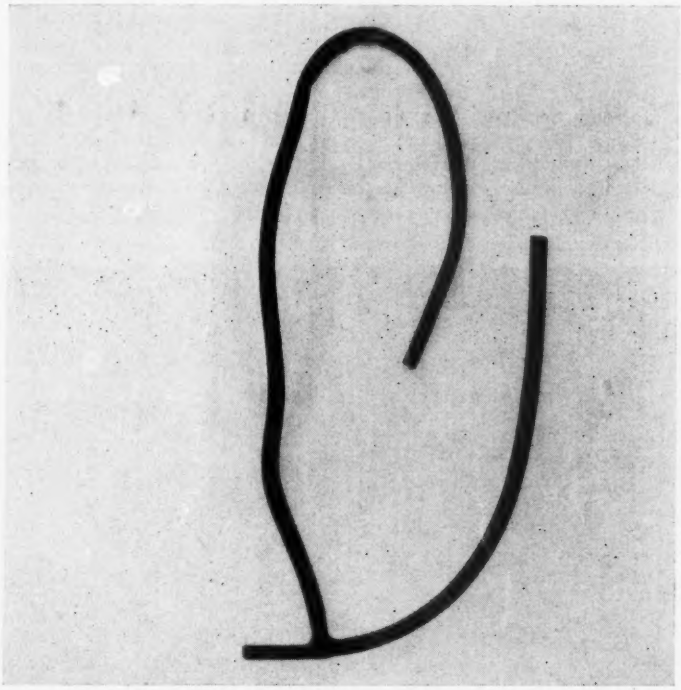

Fig. 2. T tube with one long horizontal limb. This end of the tube is placed in the distal portion of the common duct and enters the duodenum.

tions present at surgery. If there has been partial stricture formation of the papilla about a stone, and the papilla has been divided, the tube should be left as a mold for perhaps as long as three to six months, or even longer. This will aid in the prevention of a new stricture. The tube should be left a minimum of eight weeks in all cases. If reoperation becomes necessary, it is easier to carry out after a lapse of 8 to 10 weeks between sessions. The tissues are so vascular and friable prior to this interval that the technical aspects of the procedure are made quite formidable. Apparently no harm

comes from leaving these tubes for great lengths of time provided they are irrigated and provided the flow of bile is stimulated by a cholagogue. These tubes are little or no more difficult to remove than the ordinary type of T tube. Biliary drainage should cease from within 24 to 48 hours after their removal.

Illustrative Cases

Case 1. Mrs. S., aged 61, gave a long history of biliary colic with intermittent jaundice. Exploration revealed stones in the gallbladder and numerous stones in the common duct. The patency of the papilla could not be definitely established. For this reason the duodenum was mobilized and opened in a horizontal manner. Inspection of the papilla revealed a stone partially eroding its way through into the duodenum. The duodenal mucosa in this area was incised, freeing the stone. A probe was passed from the duodenal opening of the duct into the supraduodenal incision, and a long Carrel type of small rubber tube drawn into the duodenum and brought out through the incision in the supraduodenal portion of the duct. This case illustrates the most useful indication of this method of exploration of the extra-hepatic biliary duct system.

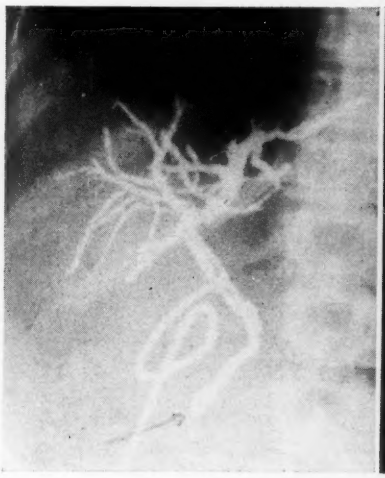

Fig. 3. Postoperative cholangiogram in Case 3. There is an obstruction in the distal end of the common duct.

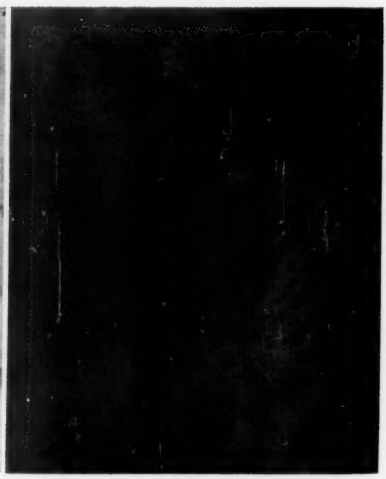

Fig. 4. Cholangiogram one week later in Case 3. The obstruction persists. It is interesting to note the dilatation of the intrahepatic as well as the extrahepatic duct system.

Case 2. Mr. R. B., a 43 year old white male, with multiple sclerosis, was admitted with a deep obstructive type of jaundice. At exploration the gallbladder and the extra-hepatic biliary duct system were found to be markedly dilated. A soft mass could be palpated in the region of the papilla. Combined supraduodenal and transduodenal exploration of the common duct was carried out which revealed a polypoid growth at the papilla of Vater. Local excision of this lesion was performed with reimplantation of the ducts into the duo-

denum about a Carrel type of tube which traversed the duodenum, the common duct, and was brought out through the latter near the cystic duct. More radical surgery was not elected due to his poor general condition and his poor ultimate outlook from the multiple sclerosis. He has subsequently remained well. The pathological diagnosis was that of adenocarcinoma, grade 2.

CASE 3. Mrs. V. G., a colored female, aged 60, had been explored eight weeks prior to the time we saw her, cholecystectomy and choledocholithotomy being done. The patency of the papilla could not be established definitely at that time. The patient's general condition was such that further surgery was not undertaken. A small T tube was placed in the common duct and the abdomen closed without further manipulation. Figure 3 represents the first postoperative cholangiogram. Note the failure of the radio-opaque substance to pass into the duodenum. Figure 4 represents another postoperative cholangiogram made approximately one week after the first one. Note the marked dilatation of the duct system, both extrahepatic and intrahepatic, and the semilunar defect in the region of the pancreatic portion of the duct. At the second operation the duodenum was mobilized, but with some difficulty, due to numerous adhesions, and combined supraduodenal and transduodenal exploration of the common duct was undertaken. The probe could not be passed through the superior portion of the duct into the duodenum. In spite of very close inspection it was quite difficult to locate the papilla of Vater. Finally, a small amount of saline solution was forced through and could be seen trickling into the duodenum. A probe was passed through this portion of the duct and on up into the incision in the common duct. The long limb of a Cattell type of T tube was tied to this probe and was gently drawn into the region of the duodenum. An obstruction was met just above the outlet of the duct into the duodenum. The papilla was incised revealing a stricture about a small, remaining common duct stone. The duodenum was closed about the long limb of the T tube and the vertical limb was brought out through the supraduodenal portion of the duct. An interesting observation in this patient was that a probe had almost made a false passage into the duodenum near the papilla. This patient had a benign stricture of the distal end of the duct, undoubtedly due to fibrosis, as a result of an inflammatory process about an impacted stone in this area.

CASE 4. Mrs. C. H., a diabetic, 54 year old white female, had an external biliary fistula of eight years' duration secondary to cholecystostomy for stones. Combined exploration of the duct was carried out, at which time a partial stricture at the distal end of the duct was revealed. This was divided and a small stone was discovered and removed. The long limb of a Cattell tube was placed in the duct and the duodenum carefully closed. Her postoperative course was uneventful. The tube was removed in eight weeks and she has subsequently remained well.

COMMENT

This method of exploration of the common duct is undoubtedly of considerable value when there is doubt in the surgeon's mind as to whether or not the papilla of Vater has been traversed by the examining scoop, probe or catheter. It has probably not been employed more often in the past due to the fear of peritonitis, or of a fistula which might arise from opening the duodenum. These se-

rious complications can be avoided; first, by antibiotic therapy, and second, by careful mobilization permitting adequate exposure, so that the duodenum can be very carefully closed. In the presence of stones in the duct, and when there is doubt in the surgeon's mind as to whether or not those in the pancreatic portion have been removed, he should unhesitatingly mobilize the duodenum and open it in order to establish definitely the complete patency of the duct system. This should be done at the primary operation in that it is infinitely easier to carry it out at this time than at a secondary operation at a later date, at which time the normal anatomical landmarks may be greatly distorted by adhesions and the operative procedure made difficult and hazardous by very vascular, friable adhesions. It must be emphasized that this operation must not be done unless the surgeon is able to mobilize the duodenum widely so that adequate exposure for the exploration and subsequent duodenal closure may be had.

REFERENCES

1. Mahorner, H.: Combined supraduodenal and transduodenal exploration of common bile duct, Ann. Surg. *129*:766 (June) 1949.
2. Cattell, R. B.: Use of long T tube in surgery of biliary tract, S. Clin. North America *28*:659 (June) 1948.
3. Buxton, R. W., and Burk, L. B., Jr.: Choledochotomy, Surgery *23*:760 (May) 1948.
4. Orr, T. G.: Operations of General Surgery, Philadelphia, W. B. Saunders Company, 1944.
5. Welborn, M. B.: Comments on diagnosis and management of gallbladder disease, Kentucky M. J. *44*:199 (June) 1946.

COMMON BENIGN BREAST LESIONS

CLYDE C. SPARKS, M.D.
Ashland, Ky.

THE opening paragraph of a paper by Snyder, Snyder and Grosjean, appearing in THE SOUTHERN SURGEON of June, 1950, stated: "The diagnosis of benign lesions of the breast is important in order to rule out carcinoma, to avoid unnecessary or unnecessarily extensive surgery for a benign lesion, to determine the proper therapy for the breast lesion, and in some cases to establish the diagnosis of tumor or hyperplasia of the ovary, testis, adrenal cortex or pituitary."

Gibbon in reference to masses other than benign adenomata of the breast stated, "Any other mass in the breast of a woman in the neighborhood of 40 years or beyond was considered by some, at least, a sufficient warrant for removal of the whole breast. . . . Such an attitude is but an acknowledgment of ignorance, and brings discredit on surgery."

While we are quite sure we are not following the practices alluded to in the latter paragraph, it is apparent that in order to have arrived at the full concept of the first paragraph, the operative sacrifice of some nonmalignant breasts, and many other errors have been made.

This particular subject has repeatedly been discussed and therefore at times will be considered rather boring, but when we review in our minds the psychic reactions manifested by women who present themselves with breast lesions, one has little difficulty in accepting the importance of this problem. Mental responses range from the occasional stoical patient with the attitude that "I have a lump in my breast and that is that," to the more frequent complaint of a mass, or painful breasts, coupled with obvious terror.

These patients are coming for treatment earlier in their illness, and at a much younger age, largely because of the dissemination of cancer information by medical personnel and the American Cancer Society. These facts make differential diagnostic knowledge for all medical people mandatory. This may well be illustrated by comparative studies of patients in two separate four-year periods of time, as shown in table 1.

In a series of 80 consecutive cases of female breast lesions which

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Presented during the Hollywood Assembly of The Southeastern Surgical Congress, Hollywood, Fla., April 11-14, 1951.

were treated surgically in a small, general hospital, the distribution of pathological diagnosis is shown in table 2.

TABLE 1

	1937-1941	1946-1950
Average age	51	36*
Duration of symptoms	24 months	36 months**
Benign	20%	80%

*Two cases of age 66 increased this average almost one year.

**If 2 cases with 10 year histories are deleted the average would be 6.2 months.

TABLE 2

Chronic Cystic Mastitis	24
A. Cystic disease	19
1. With circumscribed abscess	1
B. Adenosis	5
Fibroadenoma	15
Acute and Chronic infectious mastitis	6
Intracystic papilloma	2
Lipoma	1
Sweat gland tumor	1
No pathology	1
Malignancies	30

During this period of time some 50 cases of clinically nonmalignant breast lesions were not submitted to surgery, and the diagnosis showed essentially the same ratio of adenosis and cystic disease as those treated surgically. Also a 20 per cent ratio of mastodynia was noted. One case of virginal hypertrophy, and 2 cases of pubescent mammary asymmetry with hyperplasia were observed.

The type surgery performed in the benign lesions listed in table 2 is divided into operative procedures as shown in table 3.

TABLE 3

Partial mastectomy	28
Removal of tumor (enucleation)	18
Simple mastectomy	1
Radical mastectomy	1
Incision and drainage	2

Partial mastectomy is used here to describe the removal of more than the mass itself and less than the entire breast. The radical mastectomy was an error. Subsequent tissue studies revealed a chronic fibrous cystic mastitis with some lymphadenitis. For the

most part three types of incisions were used in these cases: either a curved incision along the areola, a radial incision, or a Warren incision. These were chosen according to type and location of the breast lesion.

The complaints as registered in this series of cases treated by operative surgery are as indicated in table 4.

TABLE 4

Mass without pain	19
Mass with pain	21
Pain alone	4
Mass with pain and discharge	1
Discharge or bleeding	1
Recent growth	10

Variations in the menses are not uncommon in these breast lesions, and their incidence is shown in table 5.

TABLE 5

Abnormal as to frequency	20%
Abnormal as to duration	15%
Post menopausal	8%

The number of pregnancies occurring in this group shows in table 6 the following percentage ratio:

TABLE 6

No pregnancies	20%
One pregnancy	31%
Two or more pregnancies	49%

For sake of discussion of the groups of benign lesions found in our series, we may divide these tumefactions and abnormal enlargements as follows:

TABLE 7

I. The hypertrophic mammary gland changes.
   A. Mammary asymmetry at puberty with hyperplasia.
   B. Virginal hypertrophy.
II. Mammary Dysplasia.
   A. Mastodynia.
   B. Chronic cystic mastitis.
      1. Adenosis.
      2. Cystic diseases.

III. Benign neoplasms of the breast.
A. Fibroadenoma.
B. Intraductal or intracystic papilloma.
C. Sweat Gland Tumor.
D. Lipoma.

For the sake of completeness in discussion we should add infantile hypertrophy, and the several benign non-indigeneous tumors of the breast. These benign neoplasms may arise from any tissue present, and the infantile hypertrophy is mentioned, as it calls immediately to mind the possibility of adrenal cortex, granulosal cell tumor or cyst, teratoma or chorio-epitheliomas of the ovaries, and occasionally third ventricle lesions.

Mammary asymmetry at puberty was seen twice in this series. One case at age 8, and 1 at age 9. In both cases the opposite breast was completely infantile and no subareolar "button" was felt. The involved breast approached normal adult size. Pain was the chief complaint and preceded the enlargement. The growth had been rather gradual in the younger patient for four months and in the older girl for five months. The enlargement had been stationary for one and two months respectively. In both instances some tenderness was noted. This response of one breast over the other to estrogenic stimulation requires no treatment, and it is expected that in both of these cases relative symmetry will be established and maintained.

Virginal hypertrophy occurred in 1 case. This patient has huge breasts with wide, thin areola, short, flat nipples, mild cyanosis and some dilated superficial veins. The history indicates a period of six months of rapid growth at age 13, with pain and extreme heaviness. Scanty menses were present until age 18. She became markedly selfconscious for a period of years, but apparently her physician to whom she was finally taken has done well with her. She has made a very nice adjustment, and with properly fitted brassieres lives a rather normal life. She is married and has one child which she was unable to nurse. A definite enlargement occurred during the latter part of gestation and has remained permanent. She is fully aware of relief offered by mastectomy or plastic procedures but is well adjusted and wishes to continue in her present status. She is relatively young at the present (23), and may have more difficulty. Extreme sensitivity of the breast to hormonal influence is held as the causative factor. Amputation is probably the treatment of choice if anything is done, with plastic procedure sometimes indicated. Fortunately this condition is not a frequent one.

Mastodynia as well as some of the other lesions, such as chronic

cystic mastitis and adenosis, is a rather indefinite entity and the pathological picture varies considerably from case to case. They must be interrelated problems, and where one ends and the other begins, it is often impossible to clearly define.

Mastodynia is characterized by premenstrual pain, a tenseness and a granular feeling of the breast, most often in the outer upper quadrant and rather rarely in the subareolar or lower portion of the breast. It was bilateral in 30 per cent of the cases covered by this study. The average age was somewhat less than the over-all average of cases studied, being slightly over 28 years. Eighty per cent were married and a low pregnancy rate was noted.

The two distinct breast types noted by Geschickter and Copeland were observed and a quotation from Copeland describes this most ably, and is completely collaborated by our small series.

"1. Women in childbearing age who have a tendency toward sterility and who have not recently had a pregnancy. Breasts are usually well developed, menstrual history is normal and the disturbance in the breast comes late after maturity and is usually transient."

"2. Young childless women with small atrophic breasts. These breasts suggest a primary mammary deficiency arising at, or shortly after, adolescence. The menses are irregular and the women are frequently sterile."

The latter group are in general somewhat poorly adjusted individuals, and one must use extreme care in their management. These women seem to be easily given a breast fixation. It must be remembered that a body is attached to these breasts.

Progesterone offers, all in all, more relief than most other treatments but in some cases any form of therapy may produce poor, or even adverse results, particularly from a psychological standpoint.

Chronic cystic mastitis. This is a rather loosely used term and perhaps justly so, since the tissue examinations in this group vary greatly from breast to breast, and from section to section. Pathologists, more and more, are preferring to report the tissue changes in these breasts, rather than differentiate by named diagnosis. The clinical evaluation is therefore up to the attending surgeon.

Adenosis. Five out of 24 cases of chronic cystic mastitis were classified as adenosis, but by the very nature of the relationship of adenosis and cystic disease, an ironclad classification cannot, of course, be made and some cases show evidence of both entities. The average age was 33 years. Twenty per cent were bilateral. Twenty per cent were in normal-sized breasts and 80 per cent of these cases

had small, undersized, poorly developed breasts. Pain was the one symptom common to all cases, and the one physical finding present in all cases was a granular feeling which in 80 per cent of this particular group was in the outer, upper quadrant. Tenderness was present in 80 per cent and a history of a preceding mastodynia was noted in 40 per cent of these cases. Recent growth was noted in 40 per cent. The pathology was not different from that usually described, such as increased fibrosis, multiple small cysts sometimes with adenomas, papillomas, epithelial proliferation and intraductal hyperplasia. The close relationship of this pathological picture to malignancy of the breast makes it imperative that we have a rather thorough understanding of this common breast lesion. The frequency of cancer in this type of breast has been stated by Geschickter as five times that of a normal breast.

Endocrinologically these patients will show evidence of adolescent hyperestinism or a combined lowered corpus luteum influence. The latter group presents certain difficulties in their clinical management, since they are tense and psychologically disturbed. In management of adenosis, with a carcinoma incidence of 2 per cent, it is certainly obligatory that malignancy be ruled out. Biopsy of all cases of adenosis with a history of recent growth, or with one circumscribed mass that is larger than the other granular feeling areas is certainly indicated. If all reasonable doubt of malignancy has been removed, progesterone therapy may be of value but the results are not so striking as in mastodynia.

Cystic disease (*macro-cystic*). In the series we are reviewing this condition occurred almost four times as frequently as adenosis, but as occurred in that lesion, the pathology overlaps, and a certain number of cystic disease cases undoubtedly follow mastodynia and adenosis in sequence. Multiple small cysts are not infrequent. Seventy per cent of these cases occurred in multipara, and in the remaining 30 per cent, some long period of time had elapsed since the first pregnancy. Most of these cases showed menstrual disorders, and most were at or near the menopausal age. The average age of multiparous cases was younger. One case of cystic disease with increased fibrosis and a circumscribed abscess was present in this series.

The treatment of cystic disease is primarily surgical both from a therapeutic and diagnostic standpoint. Secondarily, endocrine therapy is of value to aid in combating estrogen excess which is probably a major causative factor. The use of endocrine preparations is directed at the prevention of recurrence. Endocrine therapy is not advised prior to an accurate diagnosis.

In our experience we have not been able to derive the benefits ascribed to transillumination by many writers.

Benign neoplasms. In the series of 50 consecutive operations for benign breast lesions, we have previously noted that 15 cases of fibroadenoma occurred. The average age in this series was 29, and the youngest was 14. Two cases were at menopause and both described recent growth. One occurred during pregnancy and in this particular case the mass enlarged rapidly in the second and third months of gestation and was promptly removed. The consistent complaints were noted as a mass, slowly growing in most cases, and some indefinite pain and tenderness. In a few cases mastodynia was noted, but it is believed to be incidental rather than as a part of the pathological process. Physical findings consisted of a firm, movable, nonfixed tumor, and in none of the cases was dimpling of the skin or retraction of the nipple noted. One case was bilateral. Treatment is excision, both for diagnosis, immediate treatment, and for prevention of fibrosarcoma and myxomata that may occur in later life. Recurrences may occur in the same or opposite breast.

Intraductal and intracystic papillomata. Two cases of this category were noted. In one a simple mastectomy was performed because of bleeding from the nipple at age 46. In the other, the papilloma was intracystic and discovered incidentally. It is worthy of note that during the same period of time covered by this group of cases, that one large intracystic and one small intraductal papilloma were found to be malignant. In 1 case a simple mastectomy was done, followed shortly by a radical mastectomy. The intracystic papillomatous malignancy was discovered by a partial mastectomy, followed later by a radical breast resection.

The bleeding nipple offers one of the most disturbing problems in handling breast complaints. Transillumination, palpation, roentgenograms with opaque media, and inspection, are of course valuable, but all of these procedures leave us completely at sea on various occasions. We must therefore in those cases depend on continued observation, exploration of breast, or simple mastectomy. The psychic stability of the patient is important in these problems but, all in all, we prefer to do a simple mastectomy in the so-called cancer age. Pathologically, here again as in previously discussed lesions, there is some overlapping as in the case of papillary hyperplasia in adenosis. It is worthy of note that for differential purposes, adenosis is usually peripherally placed, while intraductal and intracystic papillomata are centrally placed.

Other benign neoplasms of the breast appearing in this series

were one lipoma, and one sweat gland tumor. Others are described, but for the most part are of academic interest only.

Any paper referable to this subject would be at least relatively incomplete without some comment on the value and place of simple mastectomy as a procedure in our surgical armamentarium. Slaughter and Pearson have stated that this is an uncommonly indicated operation and is performed too frequently. We subscribe to this in its entirety, but wish to advise its use as a palliative procedure, in cases where carcinoma cannot after careful study be eliminated—in sarcoma, in some inflammatory diseases, in cases of diffuse and recurrent benign neoplasms with malignant tendencies, and in cases of adenosis with papillary hyperplasia.

Summary

A brief review of the present status of the common benign breast lesions has been presented with emphasis on the following points:

1. Differential diagnosis.

2. A plea for conservatism in management of nonmalignant breast lesions in as far as possible.

3. That it is of tremendous importance to remember the patient as an individual rather than the breast as a problem within itself.

4. That with proper knowledge of breast lesions a tremendous amount of useless mutilating surgery can be avoided.

5. That many breast problems present not a true clinical entity within itself, but a part of a general picture.

REFERENCES

1. Snyder, H. E.; Snyder, C. D., and Grosjean, W. A.: Benign lesions of breast, South. Surgeon *16:*555 (June) 1950.
2. Gibbon, J. H., Jr.: Benign tumors of breast, Atlantic M. J. *29:*526 (May) 1926.
3. Copeland, M. M.: Benign tumors of breast and associated conditions, South Dakota J. Med. & Pharm. *2:*116 (April) 1949.
4. Saltzstein, H. C., and Pollack, R. S.: Benign tumors of breast, J.A.M.A. *140:*997 (July 23) 1949.
5. Slaughter, D. P., and Peterson, L. W.: Indications for simple mastectomy, Surg., Gynec. & Obst. *85:*456 (Oct.) 1947.
6. Geschickter, C. F.: Diseases of the Breast, Philadelphia, J. B. Lippincott Company, 1943, chap. 12, p. 276.

VERTEBRAL OSTEOCHONDROMA

HUGH C. ILGENFRITZ, M.D.
Shreveport, La.

THE commonest benign tumor arising in bone is the osteochondroma or exostosis, which appears usually at the ends of the long bones of the extremities in the neighborhood of joints, and develops typically at the sites of attachment of tendons or ligaments. It is characteristically a tumor of young adults, slow growing and of long duration, and productive of little or no discomfort until its size and location cause interference with function. As a rule, the tumor has been present for some years before treatment is sought; in some cases, when the growth is in an inconspicuous location, many years may pass before its presence becomes apparent. In the latter case, symptoms may not result from the presence of the tumor until the patient has reached middle age. In occasional instances, an osteochondroma may become malignant, a chondrosarcoma resulting.

The typical osteochondroma consists of a mass of laminated cancellous bone, interspersed with marrow, attached to its point of origin by a bony pedicle and surrounded by a zone of cartilage covered by a thin layer of fibrous tissue, the strands of which merge indistinguishably with the fibers of the tendon attaching to the site at which the exostosis takes origin. Geschickter and Copeland,[1] in explaining the histogenesis of these tumors, state that the normal bony protuberances to which tendons attach are formed partly from cortical outgrowth of bone and partly from osseous tissue derived from precartilaginous connective tissue within the end of the tendon. Periosteum normally then grows across the bony protuberance thus formed, restraining further bone growth. Failure of this periosteal covering to develop may permit excessive formation of bone by the precartilaginous connective tissue in the end of the tendon, an exostosis resulting. Similarly, trauma may activate the precartilaginous connective tissue remaining in the tendon at its point of attachment to the bone, new bone formation occurring as a result of this stimulation, with development of an exostosis.

Although osteochondromas occur chiefly in the long bones in the vicinity of joints, they are found less commonly in the bones of the jaw, skull, hand, and foot, and rarely in other locations. Individual case reports have been published, describing the development of osteochondroma in such unusual locations as the sternum, ribs,[2]

Presented during the Hollywood Assembly of The Southeastern Surgical Congress. Hollywood, Fla., April 11-14, 1951.

clavicle, scapula, pelvis, and vertebral column. Geschickter and Copeland,[1] tabulating the total of 321 cases treated at the Johns Hopkins Hospital up to 1949, list 11 cases of osteochondroma of the vertebral column, 9 occurring in the lumbar region, 1 in the lower thoracic region, and 1 in an unspecified location in the cervical spine. Simpson[3] has reported a case of Horner's syndrome caused by osteochondroma of the first rib, and Moore[4] has described a clavicular osteochondroma with secondary brachial paralysis.

The symptoms produced by osteochondroma are almost entirely those resulting from pressure on neighboring structures and from interference with function by reason of the location and mass of the tumor. The diagnosis is ordinarily obvious and the appearance of the exostosis on x-ray is characteristic, even when the tumor has developed in one of the rarer locations. Occasionally, however, secondary symptoms resulting from pressure, and physical and roentgenographic findings may be so unusual that the diagnosis becomes difficult.

Case Report

The patient, a white male aged 52 years, entered the Physicians & Surgeons Hospital on Dec. 12, 1948, with the chief complaint of a firm swelling in the neck, diagnosed by the family physician as toxic nodular goiter. The patient stated that the swelling in the neck had been present for at least 13 years and had been increasing in size gradually and progressively. Treatment with iodine by mouth was carried out at intervals over a period of years with no noticeable benefit. No toxic effects were noted and the mass caused no particular discomfort until the present symptoms began about six months previously. These symptoms progressed until the patient became almost completely incapacitated; he stated that for the past three months he had been unable to see or to walk or to talk. At the time of admission to the hospital, he complained of double vision, of unsteadiness, stumbling, and inability to walk without support, and of clumsiness and fumbling in the use of the hands, with inability to write, to handle eating utensils, and to dress himself. He stated that he believed this to be due to muscular incoordination rather than to weakness. Difficulty in speaking also was pronounced at this time, the enunciation of words requiring considerable physical effort. Recently, the voice had become hoarse and husky, the left eyelid had shown a slight constant droop, and sweating had ceased on the left side of the face, although normal elsewhere. The patient further stated that his appetite was poor and he had developed a tendency to tire on the slightest exertion. Pain was not a prominent symptom, although constant discomfort was noted in the left side of the neck, the left shoulder, and the left arm.

Physical Examination. The patient was a well nourished, well developed man of middle age, in fair general health, well oriented and alert. Speech was slow, stumbling, and almost unintelligible, apparently because of difficulty in enunciation.

The head was normal in contour, with no areas of tenderness or anesthesia. The eyes were clear, extraocular movements were normal, and the pupils were round, regular, and normally reactive to light and accommodation. The left

pupil was miotic, and ptosis of the left upper eyelid was present. Ocular fundi were normal. No nystagmus was noted, and the visual fields were grossly normal. Hearing was normally acute bilaterally; the sense of smell was unimpaired.

A stony hard, rounded, well encapsulated mass was noted in the left lower neck anteriorly, filling the left supraclavicular fossa, and extending down beneath the left clavicle and over to the right side of the midline. The tumor appeared to be in contact with the inner aspect of the left clavicle and was rigid and immovable. The trachea was displaced far over to the right. Venous engorgement and moderate edema were noted over the left side of the neck, the left shoulder, the left upper chest anteriorly, and the left arm. The pulsation of the carotid artery was prominent on the right side of the neck but was not found on the left. Laryngoscopic examination revealed paralysis of the left vocal cord.

Chest expansion was equal and symmetrical bilaterally except for a lag in the left upper chest. Lungs were clear throughout to percussion and auscultation except for dullness and absence of breath sounds in the region of the left pulmonary apex. The heart was not enlarged, sounds were clear and of good quality, and no murmurs were noted. The blood pressure was 128/78 in each arm. The abdomen, genitalia, and rectum were essentially normal on examination.

Horner's syndrome was noted on the left. No evidence of cranial nerve damage was noted. Marked dysarthria was present, although there was no evidence of aphasia. The tongue was protruded in the midline. The grip was somewhat weak in each hand, and fumbling and coarse tremor were noted in both upper extremities, more marked on the right. The tendon reflexes were normal throughout; no abnormal reflexes were present, and muscle tonus was normal. Finger-to-finger and finger-to-nose tests were performed acceptably but somewhat uncertainly with the left hand. A slight tendency to astereognosis was present; other sensory tests were normal. The Romberg test was positive; also, the patient could not balance himself on either foot. The gait was uncoordinated and staggering and the patient was unable to stand alone or to walk without support because of inability to maintain his balance.

X-ray showed the presence of a large calcified mass extending into the left side of the neck and occupying the left upper lung field, with marked right lateral displacement of the trachea.

Laboratory Report. The laboratory tests were normal throughout at the time of both hospital admissions. On Dec. 13, 1948, blood count showed hemoglobin 14.5 Gm., erythrocytes 5,120,000, leukocytes 7,500, lymphocytes 22 per cent, monocytes 1 per cent, neutrophiles 73 per cent, and eosinophiles 4 per cent. Serum calcium was 7.7 mg. per cent, and serum inorganic phosphate was 3.6 mg. per cent. Serum alkaline phosphatase measured 1 Bodansky unit. Serum protein was 6.5 Gm. per 100 cc., with albumin 3.5 Gm. per 100 cc. and globulin 3.0 Gm. per 100 cc. Serum cholesterol was 231 mg. per cent and the blood Kahn and Kolmer were negative. Basal metabolism rate was —7 per cent. The urine was normal. A diagnostic spinal puncture was made in the lower lumbar region and the spinal fluid pressure was found to be 90 mm. Queckenstedt test was normal. Spinal fluid showed no cells and no increase in globulin, with protein of 54 mg. per cent. Spinal fluid Kolmer was negative and the colloidal gold curve was normal.

On the basis of these findings, a diagnosis was made of calcifying nontoxic

nodular goiter, with substernal extension. No conclusion was reached at this time as to the cause of the muscular incoordination.

Operation was performed on Dec. 17, 1948, with intratracheal anesthesia. The usual thyroid incision was made and the thyroid gland was exposed and was found to be entirely normal. The trachea and larynx were displaced far to the right by a stony hard smooth rounded encapsulated mass lateral and deep to the left lobe of the thyroid. The left common carotid artery and left vagus nerve were stretched tightly over the surface of the mass, the artery being collapsed and empty. The mass was immovably attached to the vertebral column and was closely applied to the under surface of the left clavicle. Biopsy of the tumor was taken and the incision was closed. Convalescence was uneventful.

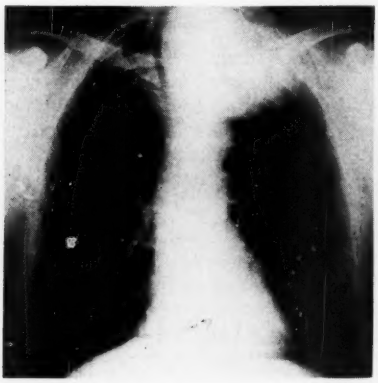

Fig. 1. Preoperative roentgenogram.

Fig. 2. Specimen removed in its entirety at operation.

Biopsy report stated the tumor to be an osteochondroma. The patient returned to his home for supportive therapy and returned to the hospital for definitive operation in February 1949.

Second operation was performed on Feb. 28, 1949, intratracheal ether again being administered. Because of the location of the osteochondroma in the left thoracic inlet and base of the neck, the third left rib was resected subperiosteally from the sternal border to the anterior axillary line, the second and first cartilages were divided and the medial half of the left clavicle was resected subperiosteally. The surface of the tumor was noted to be in contact with the inner surface of the first left rib and the upper margin of the left clavicle, the left carotid and subclavian arteries and the left innominate vein being tightly compressed between the two bony surfaces. When the first costal cartilage was divided, the collapsed vessels instantly filled with blood and the radial pulse on the left promptly became stronger. The tumor was found to extend across the bodies of the lower cervical vertebrae and into the right side of the neck, displacing the larynx, trachea, and esophagus upward and to the right, with considerable compression of these structures. The tumor was freed on all aspects from the adherent apex of the left lung below and the tissues of the neck above until the bony attachment to the ventral surface of the trans-

verse process of the seventh cervical vertebra was reached. Division was made at this point by means of a chisel, and the area of attachment was cleaned with a rongeur. A very large dead space remained in the lower neck and upper left chest. The cavity was flushed with normal salt solution and all loose spicules of bone were removed. A cigarette drain was inserted into the mediastinum posterior to the innominate vein on the left and was brought out above the clavicle. A rubber catheter was passed through the seventh left interspace in the posterior axillary line and a similar tube was brought through the second left interspace anteriorly. The bed of the resected portion of the clavicle was filled with bone chips and the incision was closed in the usual manner.

Pathological Report (Dr. W. R. Mathews). "The specimen measures 12 cm. by 5 cm. by 6.5 cm., covered by a membranous layer measuring 1 to 3 mm.

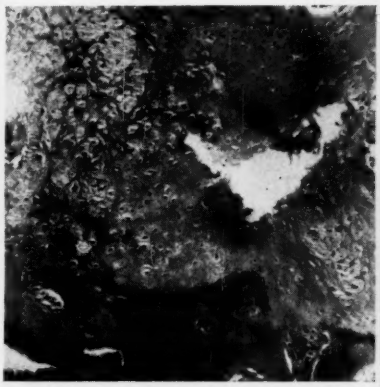

Fig. 3. Photomicrograph of representative area, showing typical features of osteochondroma.

Fig. 4. Four months postoperative. Some pleural thickening remains in left lower chest.

in thickness, with a nodular contour, and showing evidence of adhesion or fixation. The covering layer is absent on the undersurface, where numerous irregular nodules of tumor tissue are seen within the mass. These present the general appearance of a stalactite type of formation. In the center of the mass, viewed from the undersurface, there is a considerable irregular bony area measuring about 8 cm. by 4 cm. by 5 cm. This, however, is not solid bone, numerous small cavities being present through it, producing a honeycombed structure. The bone is friable here and shows a chalky appearance on the cut surface. Microscopic section shows the tumor to be an osteochondroma with areas of hyaline degeneration and myxomatous change. Some areas are clearly retrogressive in character due to chronic edema of cartilage, while others appear to represent precartilaginous connective tissue. Histologically the growth appears benign."

The routine measures of postoperative care were taken. Convalescence was satisfactory but slow, and bronchoscopic aspiration of tracheobronchial mucus was necessary on the sixth day. Following removal of the drainage tubes, thoracentesis was performed at intervals to remove accumulations of pleural fluid. The patient was discharged on March 24, 1949, in good condition. At this time the Horner's syndrome was decreasing, the left carotid and left radial pulsations were approximately normal, and the edema of the

left arm was beginning to subside. The ataxia and apraxia noted before operation were unchanged, however, and the voice was weaker and more hoarse than before operation, possibly because of the removal of the support of the tumor against the partially collapsed larynx.

Three months later, with complete healing of the operative area, the patient had recovered sufficiently to dress and to feed himself and to be up and about for half of each day. The Horner's syndrome had disappeared and speech was improved, although still a hoarse whisper. At the present time he has apparently reached maximum recovery and is fully able to care for himself, but the ataxia and apraxia, although considerably improved over the preoperative state, are still too marked to permit the patient to return to work.

Comment

The unusual location and size of the osteochondroma and its bony consistency had resulted in complete block of blood flow through the left carotid and left subclavian arteries. Collateral arterial circulation to the left arm had developed to a satisfactory degree, but the obstruction to the venous return had produced functional impairment and chronic edema. The symptoms of ataxia, general muscular incoordination and dysarthria probably were caused by impairment of the cerebral circulation with permanent neurological damage, not reversible after normal blood flow was established following removal of the tumor. The Horner's syndrome and the left arytenoid paralysis obviously were local pressure effects.

Operation was necessary as a lifesaving measure, the growth of the osteochondroma in the restricted confines of the base of the neck and the thoracic inlet having produced rapid and serious deterioration in general health, severe damage to the tissues in the vicinity of the tumor, and serious interference with the blood flow to the brain and the left arm.

Summary

A case report is presented of osteochondroma arising from the ventral surface of the left transverse process of the seventh cervical vertebra. A discussion of the symptomatology and a description of the operative procedures are given.

References

1. Geschickter, C. F., and Copeland, M. M.: Tumors of Bone, Philadelphia, The J. B. Lippincott Company, 1949.
2. Janes, R. M.: Tumors of the Thoracic Cage, Am. J. Surg. *54*:127 (Oct.) 1941.
3. Simpson, J. F.: Horner's Syndrome Due to Osteochondroma of First Rib, Canad. M.A.J. *59*:152 (Aug.) 1948.
4. Moore, M., Jr.: Clavicular Osteochondroma with Secondary Brachial Paralysis, Memphis M. J. *23*:169 (Sept.) 1948.

CONTINUOUS LUMBAR SYMPATHETIC BLOCK

David B. Corcoran, M.D., F.A.C.S.
Suffolk, Va.

In recent years the use of procaine block of the lumbar paravertebral sympathetic ganglia has been employed with increasing frequency in the treatment of conditions of the lower extremities which are due to or associated with vasospasm.

This procedure, which was first employed by Leriche[1] and popularized in this country by Ochsner and deBakey,[2] White,[3] and others, has become an important part of the armamentarium of those who treat peripheral vascular disease. It has become our most effective means of breaking up the vicious cycle of vasospasm, pain, and more vasospasm, which is so often the most serious phase of peripheral vascular conditions.

Its chief disadvantage has been the fact that in practically all cases where this block is being used, it has to be repeated at frequent intervals. Since its administration is always associated with some, and often with considerable discomfort, this necessary repetition has produced strenuous objections on the part of many patients receiving treatment. A few have been so upset by the discomfort of the first block that subsequent ones have been refused. In an attempt to overcome this difficulty, Thomason and Moretz[4] have developed a technic by which it is possible to administer a continuous paravertebral lumbar sympathetic block. They reported good results and to date have used this method on over 60 patients without significant complications.[5] We have been greatly impressed with this improvement and have employed it with good results in seventeen blocks on 16 cases. We are convinced of its effectiveness and are presenting this report in the hope that it may be brought to the attention of those who will be able to test it in larger series. Only in this manner can the value of the method be definitely established.

In any technical procedure which should have wide and frequent use, simplicity of equipment and ease of application are important. These requisites are present in the procedure worked out by Thomason and Moretz[4] to such an extent that one wonders why this technic had not been devised earlier.

The principle of continuous or fractional administration of an anesthetic agent through an indwelling needle, which was applied to continuous spinal anesthesia by Lemmon[6] in 1940 had been

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Presented during the Hollywood Assembly of The Southeastern Surgical Congress, Hollywood, Fla., April 11-14, 1951.

adapted to continuous caudal anesthesia by Hingson, Southworth and Edwards[7,8] by 1942. By 1943 Adams, Lundy and Seldon[9] had devised a technic in which an indwelling catheter was substituted for the indwelling needle and this improvement was rapidly adapted to continuous spinal anesthesia by Touhy. That this procedure could easily be applied to paravertebral block, or for that matter, to any block where prolonged action of the anesthetic agent is desirable, should have been obvious; yet this application was not reported until the article of the two authors[4] mentioned above was published in October 1949.

Equipment

The equipment we have used is identical with that employed in the original technic of Thomason and Moretz[4] and consists of a 16 gage Touhy needle with a Huber directional point, a 3½ gage Touhy catheter, a 23 gage needle with a blunt end and a sterile test tube. These catheters, if divided in half, will still be long enough for this procedure. We have not used a plug for the 23 gage needle and have not noticed any reflux of the anesthetic agent. The catheters were prepared by soaking in zephiran chloride 1:100 for 20 minutes.

Dr. Moretz is now using a specially designed 17 gage needle and a smaller caliber polyethylene tubing.[5] We tried to use this tubing, but found that it did not possess sufficient resistance to enable one to push it through the curved Huber tip when the needle was in place. If it can be employed satisfactorily with this smaller needle, the improvement will be a worthwhile one.

Technic

The patient is placed on the edge of the bed, lying on the side opposite the one to be injected (fig. 1). The spinous process of the first lumbar vertebra is identified by a superficial transverse scratch mark. A skin wheal is made 4 cm. or two and one-half finger-breadths lateral to this point. This wheal will be over the transverse process of the second lumbar vertebra. A Touhy needle is inserted through this wheal until contact is made with the transverse process. The point is then lowered and advanced for a short distance beneath the transverse process. The Huber directional needle point is kept pointed toward the midline. The catheter is then inserted through the needle until its tip is felt to slip through the curved needle point. While the catheter is held in place, the needle is removed by sliding it back over the catheter (fig. 2). The importance of not letting the Huber point be directed cephalad to any extent, and of not pushing the catheter any further after its tip

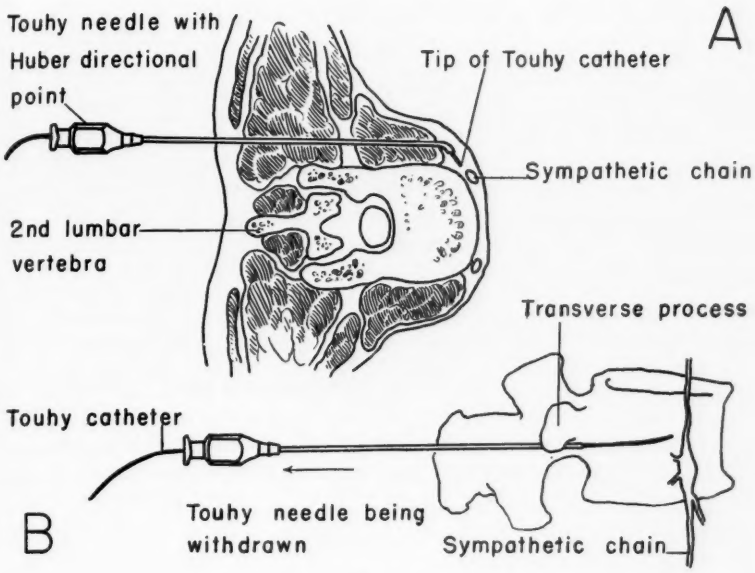
Touhy needle with
Huber directional
point
A
Tip of Touhy catheter
Sympathetic chain
2nd lumbar
vertebra
Transverse process
Touhy catheter
B
Touhy needle being
withdrawn
Sympathetic chain

Fig. 1

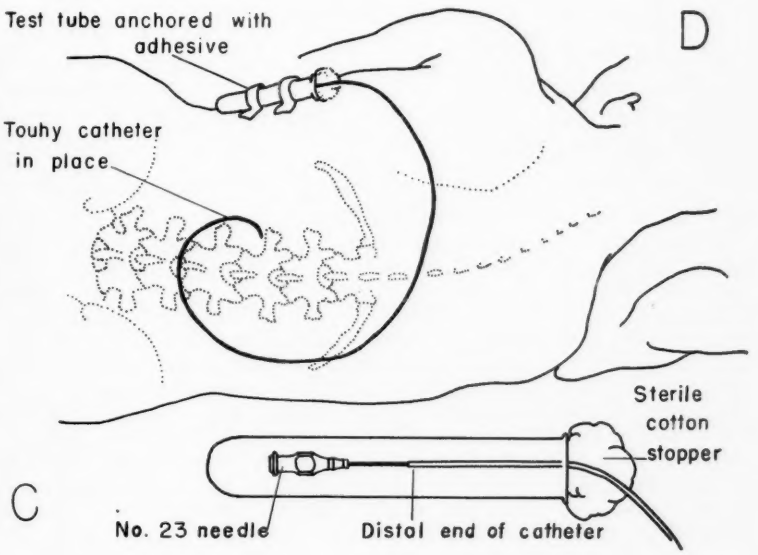
Test tube anchored with
adhesive
D
Touhy catheter
in place
Sterile
cotton
stopper
C
No. 23 needle
Distal end of catheter

Fig. 2

has been felt to slip through the point of the needle, will be emphasized later in discussing the one complication which occurred in this series. With the catheter in place (fig. 3a) a 23 gage blunt tipped needle is inserted in its free end and 8 to 10 cc. of 1 per cent procaine is injected. A small gauze sponge is then placed just beside the point where the catheter makes its exit through the skin and the catheter is folded down over this and fixed to the skin with a five inch piece of adhesive. A sterile test tube is then fixed to the side of the patient. The free catheter end and needle are placed in this tube and its mouth is tightly sealed with a pledget of sterile cotton.

The patient is encouraged to move about in bed as much as possible and will usually find that he can move the affected leg with much more facility and comfort than before the block was administered.

To minimize the possibility of infection around the catheter, which usually has to be left in situ for several days, we have followed the lead of Thomason and Moretz[4] and added 100,000 units of penicillin to each ounce of 1 per cent novocaine used. The employment of this precaution should in no way diminish the importance of maintaining a rigid aseptic technic throughout the procedure. Injections were repeated every four to five hours for the first 24 hours and every eight hours for the next 24 hours unless pain recurred after a shorter interval.

Subsequently the patient was told to inform the nurse when pain returned to the extremity and injections were repeated as often as this occurred. When edema had subsided and the patient had gone 48 hours without a return of pain, a final injection was administered and the catheter removed.

Cases

In case 3 examination of the amputation specimen revealed that the circular area of third degree burn, which formed a straplike eschar around the right leg in its lower third, extended all the way through to the bone, completely occluding all the vessels. Satisfactory block was obtained down to the circular eschar. No effect whatever was obtained below it. With this patient the area through which the Touhy needle would ordinarily be introduced, was involved in second degree burn. The block was produced by inserting a straight 15 gage spinal needle at a point about 1½ inches lateral to the usual point of insertion and advancing this needle at a 45 degree angle until contact with the body of the second lumbar vertebra was obtained. The needle point was then gently worked around the surface of the body of the vertebra until its anterior surface was reached. The stylet was then removed, the catheter inserted, and the needle withdrawn.

Case 5 provided the only complication encountered in this series. This pa-

tient was suffering from a thrombosis of the long saphenous vein of two weeks' duration.

Physical examination revealed considerable ecchymosis over the medias aspect of the left calf and ankle and a moderate amount of persistent pain. Minimal edema was present. Both the edema and pain became more severe after the patient had been up on her feet for fairly long periods.

Continuous block was chosen as a means of breaking up the vasospastic reflex and relieving the pain. The Touhy needle was inserted in the usual manner. The catheter was then introduced, and after it had slipped through the point of the needle, it was advanced an additional 2 or 3 inches. The patient then complained of severe pain in the left chest and of difficulty in breathing. Inspection of the needle revealed that the Huber directional point was rotated about 15 degrees upward from a true horizontal position. The needle was withdrawn over the catheter and the catheter itself was withdrawn 1 inch. Eight cc. of 1 per cent novocaine was injected and a satisfactory block was obtained, in that there was relief of pain and a fair temperature response.

Examination of the chest revealed hyperresonance and diminished breath sounds on the left side and a portable x-ray revealed a pneumothorax with about 25 per cent collapse. The x-ray film revealed that the catheter had taken an upward course when it was pushed beyond the needle point. Since we could not determine with certainty whether the catheter tip was within the pleural cavity, it was withdrawn another 2 inches; 10 more cc. of 1 per cent novocaine were injected, and the catheter removed six hours after it had been inserted. This patient had a return of vasospastic pain 24 hours later, but it was less severe and she refused to submit to another block. The pneumothorax cleared up spontaneously.

This case emphasized the necessity of rigidly adhering to two points in the technic described above. First, with the needle in place one must be certain that the Huber directional point is directed medially and is either exactly horizontal or pointed slightly caudad. Second, once the tip of the catheter passes through the point of the needle (this passage can be very easily felt), it must not be advanced any further.

The anatomical investigations of Melnikoff,[10] which were used by Ochsner and Graves[11] in devising a posterior approach for drainage of subphrenic abscess, have shown that though the level of the costophrenic angle of the pleura may vary considerably, it never extends below the level of the spinous process of the first lumbar vertebra. This is the level of the transverse process of the second lumbar vertebra. Therefore, the pleura will never be injured provided the catheter tip is not allowed to ascend above this level.

In case 12 we tried using a catheter which had been used before. A great deal of pressure was necessary to inject the solution after this catheter had been placed in position. There was immediate improved mobility of the involved leg, and it felt less tight. At the next injection the catheter was completely blocked and had to be replaced with a new one.

These catheters have a woven body and an external coating of shellac. After

TABLE I. SUMMARY OF CASES

Case No.	Hospital Number	Age Years	Diagnosis	Duration of Disease Before Block	Duration of Block	Results of Block	Complications
1	39865	57	Arteriosclerotic gangrene, left foot. Diabetes.	one week	5 days	Good temperature response, pain relieved. Gangrene progressive. Amputation required. Eventual fatality.	None
2	39950	38	Acute myeloid leukemia. Thrombophlebitis, both legs.	Rt. leg—3 days Lft. leg—1 day	Rt.—8 days Lft.—4 days	Good temperature response with relief of pain both sides. Edema well controlled both sides.	None
3	40144	26	Severe generalized burns. Deep circular eschar involving skin and soft tissues, L/3 right leg.	24 hours	3 days	Good temperature response down to eschar. No effect below eschar. Amputation required.	None
4	41676	45	Acute appendicitis. Acute thrombophlebitis, right leg.	3 days	6 days	Good temperature response. Definite diminution of pain. No serious edema.	None
5	40443	39	Superficial thrombosis, left saphenous vein.	2 days	6 hours	Relief of pain. Fair temperature response. Minimal edema.	Pneumothorax
6	40454	25	Acute thrombophlebitis, right leg.	2 weeks	6 days	Relief of pain, no temperature response. Minimal edema.	None
7	40468	18	Acute thrombophlebitis, left leg.	12 hours	8 days	Partial relief of pain. Good temperature response appeared on second day. Edema well controlled.	None
8	40802	63	Bronchopneumonia. Acute thrombophlebitis, right leg.	24 hours	9 days	Good temperature response. No pain at onset. Very little during course of disease. Edema well controlled.	None
9	41364	66	Diabetes mellitus. Diabetic gangrene, right foot.	7 days	6 days	Good temperature response. Definite diminution of pain and swelling. Able to move foot about with less difficulty. Eventual amputation, middle third leg.	

TABLE I. SUMMARY OF CASES (*Cont.*)

Case No.	Hospital Number	Age Years	Diagnosis	Duration of Disease Before Block	Duration of Block	Results of Block	Complications
10	41530	81	Ischemia, right foot due to arteriosclerosis. (Pain and sensation of coldness, right great toe. Right foot cold.)	2 days	4 days	Immediate diminution of pain. Patient stated that foot felt warmer. No definite palpable temperature change. Developed increase in temperature next day.	None
11	41551	74	Varicose ulcer, left leg with painful cellulitis.	2 weeks	4 days	Definite diminution of pain. Good temperature response.	None
12	41713	63	Diabetes mellitus. Embolus, left popliteal artery.	2-3 hours	5 days	Immediate improvement in ability to move leg about. Leg felt "less tight." Catheter re-inserted because of clogging. Increase in temperature of left calf and knee within 20 minutes. No change in foot. Patient eventually developed gangrene and supracondylar amputation was necessary.	Had to re-insert catheter. (Used before).
13	41848	57	Thrombophlebitis, left.	12 hours	4 days	Good temperature response. Diminution of pain. Diminution of feeling of tightness. Able to move left leg better.	None
14	41921	47	Diabetes. Cellulitis of left great toe.	1 week	4 days	Good temperature response after 24 hours. Patient did not have any pain. Rapid reduction of swelling and resolution of cellulitis.	None
15	42328	33	Compound fracture with severe crushing injury, left leg.	12 hours	3 days	Fair temperature response. Diminution of pain and sensation of tightness.	None
16	42498	67	Diabetes. Arteriosclerosis. Cellulitis, right foot with circulatory impairment.	2 days	4 days	Fair temperature response. Sensation of warmth and diminution of feeling of tightness. Diminution of pain.	None

they have been left in place for several days further use is not advisable. In addition to becoming blocked, when they are used a second time, the external coating is apt to crack, allowing the catheter to leak.

Blocking of a new catheter due to kinking may be produced if, during insertion, forward progress is not stopped immediately after the tip of the catheter is felt to pass through the Huber directional tip of the Touhy needle.

Discussion

A satisfactory block was achieved in all the cases in this series. As can be seen from table I, in all cases there was definite diminution or disappearance of pain. In most cases this relief lasted four or five hours at first. This corresponded to the findings of White,[3] who reported that a procaine block produces the effects of a sympathetic ganglionectomy for from two to four hours. These findings were substantiated by Thomason and Moretz[4] who found that the maximum elevation of skin temperature lasted two and one-half to three hours after the block and at the end of that time began decreasing toward the pre-block level.

Temperature response finding was not as uniform as diminution of pain. Thermocouple measurements were not made, but in each case in which a satisfactory temperature response was recorded, this testimony was obtained from the patient and from two independent observers who did not know which leg was being treated when they made their decision.

It is interesting to note that 2 of the patients who did not have discernible skin temperature changes voluntarily mentioned that the leg felt warmer. There did not seem to be any relationship between degree of skin temperature change and degree of pain abolition, some of the most marked temperature responses being associated with only average pain relief.

In none of these cases was edema a problem. Those which had edema of any consequence before the block was established showed definite improvement. Those in which the swelling was minimal when the block was started did not develop any significant increase.

Other subjective manifestations of an effective block were: a disappearance or a diminution of the feeling of tightness, which was usually present in the affected leg; and an ability to move the extremity about with greater ease and comfort. These manifestations were present in all of the cases except case 2.

Conclusions

There is no doubt that in some cases once the vasospastic reflexes are blocked by anesthetizing the vasomotor nerves they are not re-

established. These cases are the ones which do not require more than one injection when the single injection type of block is used. They are unfortunately few and far between. The usual response is a gradual return of pain after four to five hours. This would indicate that even daily repeated blocks are far from ideal.

Since the damage we are trying to prevent is produced by vasospasm, the most effective therapeutic approach is one which will break the vasospastic reflex and keep it broken until a point in the pathologic process is reached where it will no longer be re-established.

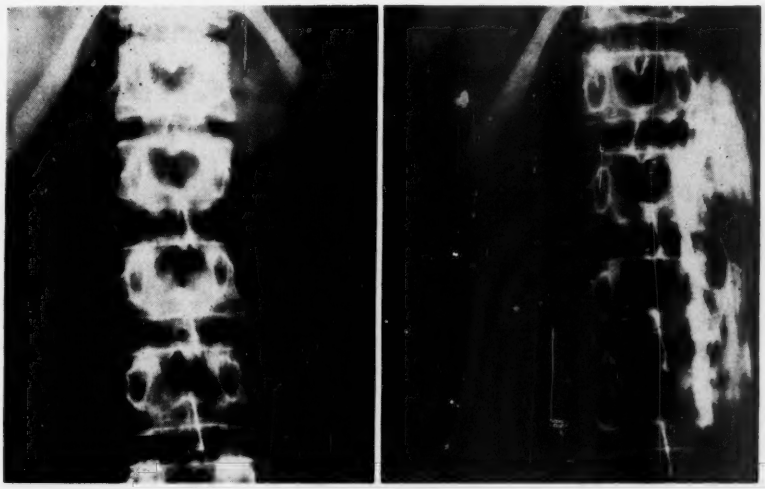

Fig. 3-a Fig. 3-b

Fig. 3. Anteroposterior views showing: a, catheter in place; b, distribution of 10 cc. of injected diodrast.

The number of cases reported is small but the results are impressive and we would like to see this method employed in larger series. We believe that it should supplant the technic of lumbar sympathetic block as it is commonly employed today. We can demonstrate both from clinical results and from the distribution of injected diodrast as visualized (fig. 3b) that in the great majority of cases it is not necessary to insert more than one needle to achieve an effective block. The technic presented here is simpler than the technics employed in the usual lumbar sympathetic block because with the use of the Huber directional point, it is not necessary to change the direction of the needle after the level of the transverse process has been reached. Any physician who is able to administer an effective lumbar sympathetic block should be able to use this method. As the

technic is employed in larger series of cases, there will probably be some failures, but these failures should not be any more frequent than those which have been encountered in the past with other methods.

The main advantage, of course, is that which accrues to the patient in that the block only has to be administered once. This is also an advantage to the physician since subsequent injections can be administered by the nurse under his supervision. The fact that the block can be repeated with such ease and can be made continuous should enhance its therapeutic effectiveness, since it has been shown that the maximum effects of the single block last only four to five hours at the most.

The continuous sympathetic block which can be obtained with this method should provide a much more efficient means of estimating the improvement which may be expected from a surgical sympathectomy in certain cases where that procedure is being considered.

If a continuous block is used as a preliminary to sympathectomy in peripheral vascular sclerosis it should help to isolate the occasional case which may be made worse by the later procedure.

In some cases of acute arterial insufficiency, it should be possible to obtain a more accurate evaluation of the adequacy of the collateral circulation after the release of vasospasm by this method than by a single or repeated blocks. In some of these cases a prolonged continuous block may render a sympathectomy unnecessary and in others it may make it possible to delay the sympathectomy until the patient is in better condition.

Thomason and Moretz[4] used this procedure in the treatment of 2 cases of painful cellulitis and obtained gratifying relief of pain. They believe that the abolition of the vasospastic reflex shortened the course of the disease. This finding deserves careful investigation. There can be little doubt that the pain which is associated with some cases of cellulitis is due to vasospasm and the abolition of this factor in addition to relieving the pain should improve the circulation to the area and prove a valuable adjunct to the accepted forms of therapy.

This technic could be employed with very little modification to any block where it would be advantageous to obtain a continuous effect.

Stellate ganglion block which is being used frequently today in the treatment of cerebral thrombosis is an outstanding example.

Summary

A method of performing and maintaining a continuous lumbar

paravertebral block has been described. The results obtained with this procedure have been presented. Its advantages have been indicated and some of its many possible applications mentioned.

REFERENCES

1. Leriche, R.: The Surgery of Pain, Baltimore, Williams and Wilkins Co., 1939.
2. Ochsner, A., and deBakey, M.: Peripheral vascular disease: critical survey of its conservative and radical treatment, Surg., Gynec. & Obst. *70:*1058 (June) 1940.
3. White, J. C.: Diagnostic blocking of sympathetic nerves to extremities with procaine, J.A.M.A. *94:*1382 (May 3) 1930.
4. Thomason, J. R., and Moretz, W. H.: Continuous lumbar paravertebral sympathetic block maintained by fractional instillation of procaine, Surg., Gynec. & Obst. *89:*447 (Oct.) 1949.
5. Moretz, W. H.: Personal communication to the author.
6. Lemmon, W. T.: Method for continuous spinal anesthesia, Ann. Surg. *111:*141 (Jan.) 1940.
7. Hingson, R. A., and Southworth, J. L.: Continuous caudal anesthesia, Am. J. Surg. *58:*93 (Oct.) 1942.
8. Hingson, R. A., and Edwards, W. B.: Continuous caudal analgesia in obstetrics, J.A.M.A. *121:*225 (Jan. 23) 1943.
9. Adams, R. C.; Lundy, J. S., and Seldon, T. H.: Technic for continuous caudal anesthesia and analgesia, S. Clin. North America *23:*1196 (Aug.) 1943.
10. Melnikoff, A.: Die Chirurgische Anatomie der Sinus Costodiaphragmaticus, Arch. f. klin. Chir. *123:*133, 1923.
11. Ochsner, A., and Graves, A. M.: Subphrenic abscess, analysis of 3,372 collected and personal cases, Ann. Surg. *98:*961 (Dec.) 1933.

FIBROSARCOMA OF THE ILEUM

Two Case Reports

HERMAN MAHAFFEY, M.D., F.A.C.S.
Louisville, Ky.

"TUMORS of the small intestine are relatively rare. Malignant tumors of the small intestine are yet more rare." These two sentences are the beginning of a case report of fibrosarcoma of the ileum which we made to the Kentucky Medical Association in Lexington, Kentucky, in September 1940. The first of the 2 cases being reported today is the same case.

Ewing states, "Malignant tumors of the small intestine form only 3 per cent of the malignant tumors found throughout the intestinal tract." In the May 1949 issue of *The American Journal of Surgery,* Irwin E. Siris[2] of New York discusses malignant tumors of the small intestine and reports 4 cases. The 4 cases which he reported are classified as follows: (1) myosarcoma of the duodenum with lymph node involvement; (2) gelatinous papillary carcinoma of jejunum; (3) sarcoma of the ileum with differential nerve and muscle stains being inconclusive; and (4) leiomyosarcoma of small intestine (anastomosis approximately 12 cm. from ileocecal valve). This case presented involvement of the sigmoid, the ileum being attached to it, and a portion of the sigmoid also was resected. Thus, 2 of the 4 malignancies of the small bowel which he reported were sarcomas of the ileum.

In this paper Siris states that, "In 1945 Shallow, Eger and Carty made an exhaustive study of intestinal malignancies among 137,174 general autopsies collected from the literature from 1858 to 1938 and tabulated 5,034 in the large and 134 in the small intestine, or an incidence of small intestinal malignancies of 0.1 per cent, or about 36 times less frequent than large intestinal carcinoma. They listed the frequency of the small intestinal malignancies among general malignancies and found that 3 per cent of intestinal carcinomas and 60 per cent of intestinal sarcomas occur in the small intestine. Of 269 cases they were able to tabulate, including 38 of their own, 186 were carcinomas and 83 were sarcomas."

In this survey Shallow, Eger and Carty found 73 carcinomas and 12 sarcomas in the duodenum, 75 carcinomas and 21 sarcomas in the jejunum, and 38 carcinomas and 50 sarcomas in the ileum. Their conclusion was that, "Carcinoma is twice as common as sarcoma and that malignancy in general occurs with equal frequency in all

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Presented during the Hollywood Assembly of The Southeastern Surgical Assembly, Hollywood, Fla., April 11-14, 1951.

three divisions of the small intestines, the ileum ranking lowest for carcinoma but highest for sarcoma."[2]

"Lymphosarcomas make up two thirds of all sarcomas of the small intestines, while leiomyosarcomas constitute one fourth of the total. Fibrosarcoma and neurofibrosarcoma rarely occur."[11]

Maxwell, Crile and Dinsmore[3] state that, "From 1922 to 1946 there occurred among our patients 40 malignant tumors in the small intestine, 36 of these being found at operation and 4 at autopsy. Of the three varieties of tumors, carcinoma occurred in 21 cases, sarcoma in 13 cases, carcinoid in 5 cases and both a sarcoma and a carcinoid in the terminal ileum in 1 case." Of the sarcomas found, 5 were in the jejunum and 6 in the ileum.

Frank, Miller and Bell[4] state that, "Sarcoma of the small intestine occurs more frequently in males. They reviewed 102 cases in 1941 and found occurrence in 75 males and 27 females." This ratio is almost 3 to 1. C. W. Mayo[5] in 1940 found the occurrence of small intestinal malignancies among men two and one-half times that among women. Our 2 cases occurred in women.

Figures tabulated show sarcoma of the small intestine to occur most often in middle life. Of the 102 cases of sarcoma tabulated by Frank, Miller and Bell,[4] 61 cases were between the ages of 30 and 60 years, with 26 of these cases being between the ages of 50 and 60 years; 35 cases being between the ages of 30 and 50 years. All of the sarcomas tabulated by Maxwell, Crile and Dinsmore[3] were in the third, fourth, fifth and sixth decades of life, 11 of the 14 cases being before the sixth decade. The age of our first patient was 52 years, and of the second patient 67 years.

We have no theory to offer as to the etiologic factor of sarcoma of the small intestine. Very little can be found in the literature covering the causative factor. However, Frank, Miller and Bell[4] made the statement that, "Trauma is thought to be a causative agent of small intestinal sarcoma." They cited references which tend to support this belief. The first of our patients gave a history of falling down some steps three weeks prior to the onset of her immediate symptoms and stated that she had not felt well since this fall. There were no signs of injury at any time following the fall.

Perforation is extremely rare. Six cases of perforation had been reported in the literature up to April 1939, according to Lewis.[6] Frank, Miller and Bell[4] found 7 cases of perforation due to sarcoma reported by Chont[7] and reported one of their own, bringing the total to 8 cases of perforation reported in 1942.

Symptoms may be varied and vague or they may be spectacular

and dramatic. They will vary depending on the type of lesion; whether it encircles the bowel, which this type of tumor does not tend to do; whether it is extrinsic or whether it protrudes into the lumen of the bowel, as it did in one of our cases; also, depending on whether intussusception, obstruction or hemorrhage is present, and depending on the location of the tumor in the abdomen. Two of these latter conditions will be demonstrated in our case reports.

The onset of symptoms may be insidious. The patient may have loss of appetite, loss of weight, generalized weakness, fatigue, dyspepsia, nausea, vomiting and vague abdominal discomfort or blood in the stool. The accidental discovery of a mass in the abdomen by the patient, especially if he or she is thin, may be the first indication of disease. On the other hand, the first signs of the disease may be manifested by intussusception, obstruction or perforation accompanied by nausea, vomiting and severe abdominal pain. Lastly, the first sign or symptom may be massive hemorrhage which manifests itself by sudden weakness, fainting and collapse accompanied by large dark bloody stools. These patients do not vomit blood except, perhaps, where the lesion is located in the duodenum. Should the tumor be large enough it may gravitate into the pelvis and due to the location there, pressure, adhesions or perforation, it may be diagnosed as a pelvic tumor. Location of the tumor in the pelvis may cause bladder disturbances and constipation. It is possible, as in our first case, to have a diagnosis of duodenal ulcer made prior to operation or a diagnosis of pelvic tumor, as in our second case.

Examination of the blood will practically always show an anemia to be present. Especially will this be true in those cases where the tumor penetrates the lumen of the intestine with a loss of blood.

X-ray examination does not always help us, probably less than 50 per cent of these tumors being demonstrated by x-ray examination. Should the tumor be large enough it may cast a soft shadow and with consideration of other signs and symptoms a diagnosis of tumor of the small intestine may be made. It is questionable whether more than fifty per cent of small intestinal tumors are diagnosed by any and all means prior to operation or autopsy. It would be impossible to say more than that a tumor of the small bowel is present. Differentiation must of necessity be left to the surgeon at the time of operation or to the pathologist after removal of the tumor.

Prognosis in any malignant condition must be guarded. How frequently do we see those cases which we think have an excellent chance of living normally for many years, only to have them die within a few months or years; and, those whom we expect to die

rather early only to have them defy all our expectations and live a relatively long life in comfort and apparent good health, and to die from other causes. Should these tumors be diagnosed early many of these patients should live several years following a successful operation. Many of these tumors, because of their type and location, are not diagnosed until late in the course of the disease and then because some complication such as perforation, obstruction or hemorrhage. These add to the gravity of the prognosis. Ten years ago it was estimated that the immediate postoperative mortality was thirty per cent. This has been reduced due to improvement in preoperative care, *i.e.,* new antibiotics, increase in availability of blood for larger and repeated transfusions prior to, during and following operation, improvement in anesthesia and operative technic. Postoperative measures include use of the oxygen tent, Miller-Abbott tube (and tubes of other makes), continued use of antibiotics and blood transfusions. Cameron,[8] in 1937, reported 3 cases of fibrosarcoma which lived eight years, 2 which lived 13 years and one which lived 20 years.

At this point I should like to say a word about the similarity of fibrosarcoma and neurofibrosarcoma. Dr. A. J. Miller,[10] of the University of Louisville, who examined some sections of the first case reported here, made the following statements in his pathological report, "There is some question as to whether the tumor has arisen from the smooth muscle of the bowel wall or the sheath cells of the nerve trunks. It makes very little difference except for the name of the tumor, for the behavior of the two is very similar." His pathological report and diagnosis is one of two in our first case report. Many others in the literature agree with him. At times it may be difficult for the pathologist to differentiate the two tumors. We have a pathological diagnosis of both types of tumors from the same specimen by different pathologists, in our first case.

According to Anderson,[9] "Fibrosarcoma is a malignant tumor tending to differentiate in the direction of fibrous connective tissue." "Fibrosarcoma appears as a rounded, lobulated tumor often appearing circumscribed or encapsulated and hard and fibrous, or soft and friable, depending on the amount of fibrous tissue which has been formed. Areas of degeneration, necrosis, myxomatous change or cyst formation may be present. The malignancy tends to be proportional to the number of mitotic nuclei and tumor giant cells, and to the scarcity of collagen fibers."[9]

"Neurogenic sarcoma may be found in distinct relation to nerves. Features suggesting neurogenous origin are (1) arrangement of the cells in definite bundles with an interlacing pattern of the herringbone type, (2) wavy, fine elongated nuclei which tend to line

up in parallel fashion to form rows (palisading) and (3) fibrils distributed in pericellular fashion. From a practical standpoint, the criteria of degree of malignancy and the prognosis are the same for neurogenic sarcoma and fibrosarcoma, varying with the number of mitoses and tumor giant cells, and the scarcity of fibers."[9]

An interesting feature of the two cases being reported is that each was referred to us by the same general practitioner, Dr. Chas. O. Neff, Louisville, eight years apart. Another feature is that each case first presented itself with a different and very serious complication.

Case Reports

Case 1. Mrs. J. C., aged 52 years, was admitted to Kentucky Baptist Hospital Sept. 3, 1939. She was seen a short time later in consultation with Dr. Charles O. Neff.

Complaint: 1, Bloody diarrhea. 2, Nausea and vomiting. 3, Generalized abdominal pain. 4, Marked weakness.

Present Illness: Began 48 hours earlier with the passing of three to four large stools, characterized by large dark blood clots. This was followed by marked nausea, griping lower abdominal pain and vomiting (vomitus at no time contained blood). Had been confined to bed since onset because of weakness, pallor and vertigo. Continued to pass three to four large grossly dark stools daily. Nausea increased. Generalized abdominal pain continued, becoming somewhat localized in the right lower quadrant.

Three weeks ago patient fell from three steps without apparent injury but had not felt well since this fall.

Past History: Mentions slight vertigo; occasional dyspnea on exertion; has not felt up to par for past four to five years; indefinite malaise, gas pains and feeling of fullness after meals; appetite good; bowels regular and has not had to take laxatives. Patient had typhoid fever at age of 12; rectal bleeding twelve years ago, which was treated by irrigations.

Family History: Noncontributory.

Physical Examination: Moderately obese female with marked pallor, weakness, appearing acutely ill. Skin very blanched; lips and conjunctiva very pale. No glandular enlargement or nodules, suggestive of lipomas, neurofibromas or other growths. Foul and acidotic odor to breath. *Head, neck* and *chest* showed no other abnormalities. *Heart,* sounds were weak, regular, no murmurs, rate 100; blood pressure 90 systolic, 38 diastolic. *Abdomen,* flat, short, obese; no muscle guarding or rigidity. Definite tenderness over normal appendix area and about 2 inches in diameter around umbilicus. Mass not palpable. *Vaginal* and *rectal* examination did not reveal any pathological conditions.

Clinical Laboratory on admission:
Urinalysis—considered normal
Blood Count:

Hemoglobin, 40 per cent	Young cells, 2
Red blood cells, 2,200,000	Staffs, 7
Index, .9	Segmented, 83
White blood cells, 21,400	Lymphocytes, 7
Total polys, 92	Endoe, 1

Hemoglobin and red blood cells varied from day to day until operation. Large dark stools continued daily through September 14. Consultation was held with Dr. Louis Frank on September 6 and the possibility of small bowel tumor was discussed. We did not think patient was bleeding from either a gastric or duodenal ulcer, as there had been no vomiting of blood. Neither did we think patient had a lesion of the colon, as all blood had been very dark.

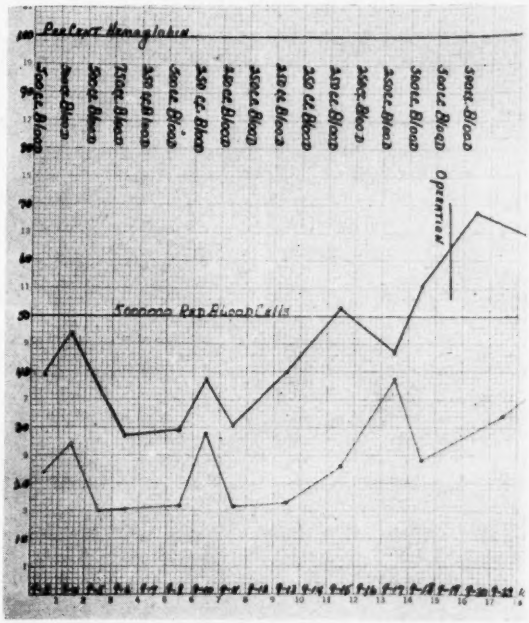

Fig. 1. Chart showing percentage of hemoglobin (based on 100%) and erythocyte (based on 5 million) variation from date of admission until after operation; also number and amount of blood transfusions.

Roentgenologic examination revealed the following: Primary film revealed the entire colon outlined by opaque material. (Patient had been receiving bismuth for some time.) There was also some of this bismuth in the small bowel and a fleck in the region of the duodenum. The primary film was otherwise negative. Fluoroscopic examination of the chest was negative. The esophagus was normal.

The stomach was hypertonic with diminished peristalsis. Considerable degree of pyloric spasm. On the greater curvature of the stomach near the duodenal bulb was a constant localized area of irritability but no definite ulcer crater. The greater curvature side of the duodenum near the pylorus is deformed. There was a constant fleck present. The descending arm of the duodenum was narrow and unusually decreased in flexibility, which was probably due to spasm and not to organic pathology.

Three and one-half hour retention study showed a stellate collection at the base of the duodenum on the greater side appearing as an ulcer. The stomach was almost empty at this time.

Twenty-four hour fluoroscopic examination showed a very small amount of barium in stomach with persisting flecking at point above described. This was again localized by a second barium meal. There was no evidence of organic lesion in the colon.

Conclusion: Duodenal ulcer near pylorus but not obstructing.

The above report was made by Drs. Keith, Keith and Shifflett.

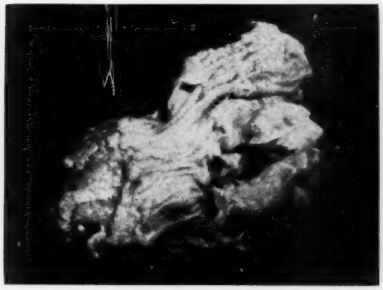

Fig. 2(a). Specimen from first case showing tumor and attached small bowel which has been opened.

Fig. 2(b). View of smooth, firm, rounded tumor from first case. A small section of tumor has been removed for microscopic examination. Section of small bowel is shown attached to each side of tumor.

The patient went to operation under spinal anesthesia on Sept. 19, 1939, with the following possible preoperative diagnosis: (1) Ulcerative colitis, (2) carcinoma of colon, (3) regional ileitis, (4) tubercular enteritis, (5) bleeding duodenal ulcer, or (6) tumor of small bowel.

Fig. 2(c). Film shows smooth, rounded tumor with protrusion of tumor into lumen of bowel in first case.

A 6 inch incision from xiphoid process down to right of umbilicus was made and abdomen was entered. Gallbladder, liver, pancreas, stomach and duo-

denum appeared normal. No ulcers of either duodenum or stomach could be palpated.

Reading the intestines, beginning at Ligament of Treitz, we encountered a small firm tumor mass approximately 2 inches in length and 1½ inches in diameter in the wall of the middle third of the ileum.

Six inches of the intestine with mesentery and tumor were resected and a lateral anastomosis was done. Appendix was removed routinely. We could not find any evidence of metastases. Complete exploration of remaining intestine did not reveal other pathological conditions. Abdomen was closed in layers. Patient received 500 cc. of blood and 500 cc. of 5 per cent glucose during operation.

Pathological Report: "Specimen consists of appendix; portion of small bowel with tumor. Gross: The appendix measures 6 cm. in length; its mucosa is smooth, gray and glistening. The tip is solid and sclerotic. The lumen is patent and filled with fecal material. In the wall of the ileum there is a soft tumor measuring 4 by 5 cm. in diameter. It extends 1 cm. into the lumen of the gut and the thin surface presents a sharply defined crater 2 cm. in diameter. On section it is grayish white in color, soft and glistening.

Fig. 3(a). Low power microscopic view showing tendency to palisading of cells; crowding of cells and hyperchromasia (dark staining) of nuclei. From specimen in 2(a), 2(b), 2(c).

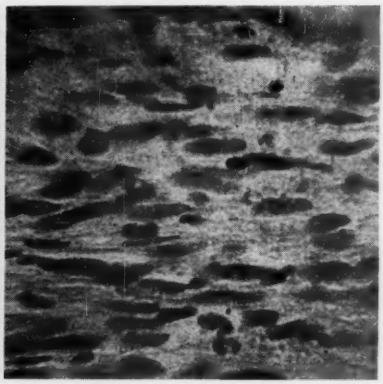

Fig. 3(b). High power microscopic view showing crowding of nuclei; pleomorphism (variation in size of nuclei). From specimen in 2(a), 2(b), 2(c).

Microscopy: Sections of the small bowel and underlying tumor mass show the mucosa atrophic. The tumor mass is composed of dense masses of cells with much stroma. Most of the cells are fairly well differentiated. There is some mitosis. In some areas fibroblasts are fairly well formed with a moderate amount of intracellular substance. There are no giant cells seen. A differentiation between a benign fibroma and a fibrosarcoma is difficult to make on the tissue. It is well encapsulated. There is no invasion of the mucosa, but in some areas the structure is too hyperplastic as to be beyond the picture of a simple fibroma.

Diagnosis: Fibrosarcoma; fibrosed appendix." (Report was made by Dr. M. Beard.)

A second report by Dr. A. J. Miller follows: "The tumor is made up of large, elongated cells, arranged in interlacing bundles. The cytoplasm is abundant and stains fairly well and there are some palisade forms. There is some question as to whether the tumor has arisen from the smooth muscle of the bowel wall or the sheath cells of the nerve trunks. It makes very little difference, except for the name of the tumor, for the behavior of the two is very similar. The tumors are apt to be multiple but recurrence after resection of the bowel is not likely. The malignancy is of a very low grade. These tumors are also quite resistant to radiation therapy. There is some tendency for malignancy to increase as time goes on. If this is the patient's only tumor, there should be no more trouble. Metastases are not expected.

My diagnosis is: Neurofibrosarcoma."

This patient made an uneventful recovery and was discharged from hospital on the fifteenth postoperative day.

Final Diagnosis: Fibrosarcoma of ileum; secondary anemia; chronic appendicitis.

On Nov. 13, 1939, the hemoglobin was 85 per cent and red blood count 4,570,000. This was considered normal.

There was a distressing sequel to this case. Twenty-six months later a kidney operation was necessary. It was performed through an abdominal incision so that abdominal contents could be explored. No signs of metastases were present. Three months following this, or 29 months following her first operation, this patient committed suicide by gunshot wound through the heart. An autopsy did not reveal any evidence of metastases in either the abdomen or chest.

CASE 2. Mrs. J. T. P., aged 67 years, was admitted to the Methodist Deaconess Hospital May 19, 1948, complaining of anorexia, nausea, vomiting and constipation. She had no bowel movement for past five days.

History: Patient stated she had had no serious illness until the present time. Has had no bloody stools.

Present Illness: Loss of appetite three weeks ago; loss of 10 pounds weight in past three weeks, nausea and vomiting for past four days and no bowel movement for past five days.

Physical Examination: White female, aged 67 years; 5 feet, 4 inches tall, and weighing approximately 120 pounds. She appeared acutely ill and in distress. Head, neck and chest were not remarkable. Fairly well developed and nourished. Blood pressure 154 systolic and 76 diastolic; pulse 104. Heart sounds were clear, distinct and regular. There was a faint systolic murmur over the mitral area. Abdomen was distended and tympanitic with some tenderness over the lower left quadrant. We were unable to palpate any masses. Vaginal examination revealed a tender, firm, fixed mass in the left pelvis. We were unable to say whether this mass was connected with female organs or in the sigmoid. We could not outline the uterus. Cervix appeared normal to palpation and inspection. Rectal examination also revealed this mass, which did not seem to be in the rectum. Sigmoidoscopic examination, by Dr. Reising, did not reveal a rectal tumor or ulcer but it did show extrensic pressure closing sigmoid just above rectum.

Laboratory Findings: Urinalysis: amber, clear, acid; S. G. 1.010. Albumin,

negative. Sugar, negative. Microscopic: Phosphates, occasional pus cell. Hematology: Hemoglobin, 53 per cent; red blood cells 2,700,000; white blood cells 13,400; polys 80 per cent; lymphocytes 13; monocytes 7.

X-ray Examination: Barium enema revealed obstruction in sigmoid area, with large amount of gas in ascending colon and small bowel.

Fig. 4. X-ray film showing almost complete obstruction of bowel in region of sigmoid. There is marked dilatation of entire colon and some dilatation of small bowel. A tumor is not visualized.

Preoperative Diagnosis: Obstruction of rectosigmoid due to pelvic tumor. Patient received 500 cc. blood transfusion day before operation and immediately after operation.

On third day following hospitalization operation was performed under spinal anesthesia supplemented by cyclo-propane gas and oxygen, given by Dr. Helm. A left paramedian incision was made approximately 6 inches in length. Exploration of upper abdomen by palpation revealed a normal gallbladder, liver, stomach, duodenum and pancreas. Small intestines were quite distended. A firm nodular mass, irregular in outline, was located in the left pelvis, adhered to the rectosigmoid, uterus and surrounding structures. This mass was freed with difficulty, and when delivered through incision was found to be a tumor originating in the wall of the ileum. The tumor was vascular, nodular, firm and grayish white in color and roughly 3 by 3 inches in size. This was the only mass present in the pelvis. While freeing adhesions between this mass and surrounding structures a small abscess was ruptured. The pus was evacuated by suction and sponges.

Approximately 8 inches of ileum, mesentery together with the tumor were resected. Ends of bowel were closed and a lateral anastomosis completed (contrary to our usual procedure) with two layers of chromic gastrointestinal catgut sutures. We were to regret not using silk or cotton for the outer layer of sutures later. Seventy-five grains of sulfanilamide were left in pelvis and around anastomosis and abdomen was closed in layers without drainage, using interrupted cotton for fascia and continuous cotton for skin.

Pathological Report: "Gross appearance—A nodular tumor mass approximately the size of an orange, reported from wall of small intestine.

Microscopic Examination: Sections show specimen is made up of atypical deeply staining young fibroblasts, many of which are ovoid in shape, others spindle with few large deeply staining nuclei and occasional deeply staining mononucleated giant cell. There is an area slightly blood stained, showing a number of apparently newly formed blood vessels and a chronic leucocytic reaction."

Diagnosis: Fibrosarcoma.

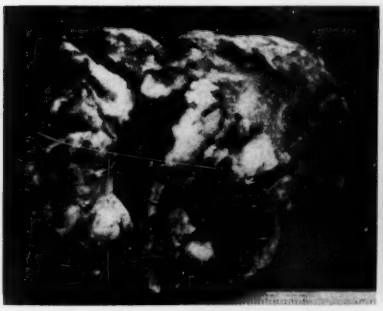

Fig. 5(a). View showing irregular, lobulated, firm, more or less rounded tumor resected together with approximately 8 inches of ileum with attached mesentery in case 2.

Fig. 5(b). Second view of tumor in case 2 showing tendency to lobulation of tumor, with attached portion of small bowel (upper left).

Fig. 5(c). View of specimen from case 2 with small bowel opened. No protrusion of tumor into lumen of bowel.

Patient was quite ill following operation, and was placed in oxygen tent; also, was given 500 cc. blood followed with 1000 cc. of 5 per cent glucose in normal saline. Forty-eight hours later she was much improved and this continued with patient being up on the fourth day.

Notation on chart seven days following operation states, "Abdomen slightly distended but soft. Dressing changed and sutures removed; wound clean and healing primary union." She had been having normal bowel movements since third postoperative day.

On the twelfth postoperative day patient began to have diarrhea, septic temperature and abdominal distention. Vaginal examination revealed a soft bulg-

ing mass in pelvis. Diagnosis of pelvic infection was made. On the sixteenth postoperative day patient was badly jaundiced; abdomen silent and distended; could get no result from enemas.

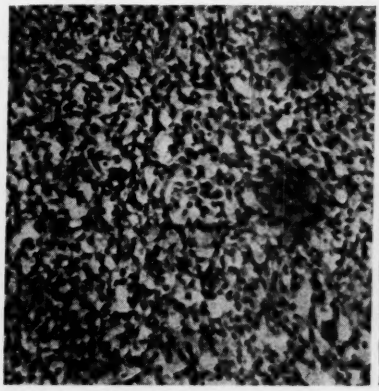

Fig. 6(a). Low power microscopic view from specimen in case 2, showing crowding of nuclei and hyperchromasia of nuclei. Palisading found in first specimen is absent in this specimen. (From specimen in case 2.)

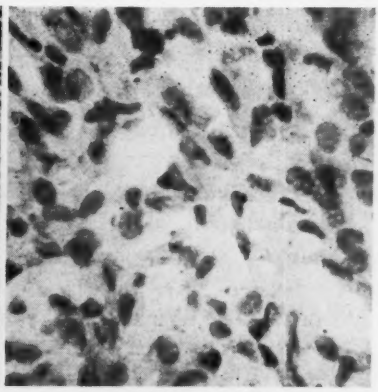

Fig. 6(b). High power microscopic view showing pleomorphism (variation in size) and hyperchromasia of nuclei. In upper left mid-portion of view a possible mitotic figure is shown. (From specimen in case 2.)

Condition of patient gradually grew worse in spite of drainage through Miller-Abbott tube, transfusions, stimulating enemas, hot packs to abdomen, and antibiotics. She developed edema over entire body, albumin, casts and bile in urine. She progressively grew worse and expired on the twenty-second postoperative day.

Permission for autopsy was obtained, abdominal only. This revealed leakage at site of anastomosis, with separation of one end of anastomosis, marked inflammation of entire large bowel, pelvic abscess, hepatitis with small abscess of liver. There was no evidence of metastasis.

Summary

1. A brief review of the literature of fibrosarcoma of ileum has been presented together with some original remarks.

2. Sarcoma of the ileum is a rare condition.

3. "Lymphosarcomas make up two thirds of all sarcomas of the small intestine, while leiomyosarcomas constitute one fourth of the total. Fibrosarcoma and neurofibrosarcoma rarely occur."

4. Two cases of fibrosarcoma of the ileum, one of which has been previously reported, are presented.

5. There is little difference between fibrosarcoma and neurofibrosarcoma. Action of the two types of tumors is for practical purposes identical.

6. Perforation due to sarcoma is extremely rare.

7. The cases presented illustrate sudden onset of symptoms; one with severe intestinal hemorrhage, and the other with intestinal obstruction.

8. Hemorrhage from the bowel must always be considered serious unless proven otherwise.

9. Frequent error in diagnosis may be made due to accompanying complications.

10. We should make every effort to localize an intestinal lesion before operation. This aids materially in the choice and type of incision.

11. Extreme care must be used in anastomosis of any portion of intestinal tract. We believe that some type of nonabsorbable suture should be used for the outer layer of the anastomosis.

Note: I wish to thank Mr. E. G. Gaslin, Jr., and Dr. John D. Allen, Jr., for their kind assistance in preparation of slides and photographs.

REFERENCES

1. Ewing, J.: Neoplastic Diseases, Philadelphia, W. B. Saunders Co., 1940.
2. Siris, I. E.: Malignant tumors of small intestine, Am. J. Surg. *77:*573 (May) 1949.
3. Maxwell, E. A.; Crile, G., Jr., and Dinsmore, R. S.: Malignant tumors of small intestine, S. Clin. North America *28:*1149 (Oct.) 1948.
4. Frank, L. W.; Miller, A. J., and Bell, J. C.: Sarcoma of small intestine: report of four cases, Ann. Surg. *115:*544 (April) 1942.
5. Mayo, C. W.: Malignancy of small intestine, West. J. Surg. *48:*403 (July) 1940.
6. Lewis, D. L.: Two cases of sarcoma of small intestine with unusual features, Brit. J. Surg. *26:*540 (Jan.) 1939.
7. Chont, L. K.: Sarcomas of small intestine and reference to their radiosensitivity, Radiology *36:*86 (Jan.) 1941.
8. Cameron, A. L.: Primary malignancy of jejunum and ileum, Tr. West. S. A. *47:*50, 1937.
9. Anderson, W. A. D.: Synopsis of Pathology, ed. 2, St. Louis, C. V. Mosby Co., 1946.
10. Miller, A. J.: Pathological report, case record of Louisville General Hospital.
11. Schafer, P. W.: Pathology in General Surgery, Chicago, Univ. of Chicago Press, 1950, p. 369.

OMPHALOCELE

Analysis of Twenty-one Cases from Charity Hospital of Louisiana at New Orleans*

H. Reichard Kahle, M.D.
New Orleans, La.

Over the last three years I have had the distinctly unusual experience of personally operating on 4 children with omphalocele and of supervising the management of a fifth child with the same condition.

The concentration of cases is in itself unusual, for omphalocele is a condition to which the word rare can properly be applied. While the reported incidence varies considerably, it is usually very low. The 7 cases reported by O'Leary and Clymer[1] in 5,197 deliveries at the University of Oklahoma Hospital over the 18 year period ending in 1941—a ratio of 1:743 deliveries—is notably high. Jarcho's[2] report of 2 cases in 5,017 deliveries at Sydenham Hospital and Hemming's[3] of 1 case in 2,035 deliveries at a hospital in Suva are also rather high. Much more typical are Kershner, Zuckerman and Kessler's[4] report of 2 cases in 20,577 deliveries over a 10 year period at the Beth-El Hospital in Brooklyn, Ludwig's[5] report of 4 cases in 20,753 deliveries in Munich, and Kern's[6] observation of 1 case in 12,374 deliveries at Sinai Hospital in Baltimore, also over a 10 year period.

My personal experience is also unusual in that 4 of the 5 children under my care left the hospital alive, owing to a concentration of fortunate circumstances. The close cooperation between the surgical and pediatric services at Charity Hospital of Louisiana in New Orleans, where these cases were managed, meant that the children were seen without delay. All the patients were treated within the last few years, which meant that all had the advantages of modern preoperative and postoperative care, including the use of whole blood and of antibiotic therapy. Finally, and most important, 3 of the 5 children had no associated major anomalies, and only in the single fatal case could the anomalies present be regarded as incompatible with life.

The interest aroused by my personal experience with these 5 cases has led me to review the records of the New Orleans Charity Hospital to study this condition further. Between Jan. 1, 1938, and Dec. 31, 1950, 21 instances of this condition, including my own 5

*From the Tulane University of Louisiana School of Medicine. Presented during the Hollywood Assembly of The Southeastern Surgical Congress, Hollywood, Fla., April 11-14, 1951.

cases, are recorded in a total of 35,444 pediatric admissions. Eight of the children were brought into the hospital shortly after birth. The other 13 were born in the hospital, in a total of 89,968 deliveries, a ratio of 1:6,921.

The investigation of these records has duplicated my experience in reviewing the records of other diseases and anomalies in young children. One senses in the older records a profound pessimism on the part of both pediatricians and surgeons when these conditions are encountered. In a 1940 record, for instance, one reads of a newborn child in good condition except for an omphalocele. The junior surgical resident, who was called in several hours after delivery, noted that he would have the visiting surgeon see the baby in the morning. The senior surgical resident, later that night, concurred. It was not until 45 hours after birth that operation was finally undertaken. There had been no preoperative preparation; the child was matched for transfusion, but got no blood. The liver could not be replaced in the abdominal cavity and death occurred three hours after operation. It is true that postmortem examination revealed defects of the heart and vascular system incompatible with prolongation of life, but that does not compensate for the spirit of resignation to the inevitable which pervades this particular record and other records of the same era. When operation was done, it was performed, as Dott[7] says, "without enthusiasm." The lack of aggressiveness is in sharp contrast to the spirit of more recent years, when, because of a more enlightened outlook on all such conditions, plus the close coordination established between the pediatric and surgical services, Dott's[7] ideal of the child's transfer from the womb to the operating table has almost been accomplished.

Another reason why the management of omphalocele has shown improvement in recent years is that the confusion which formerly surrounded it has been almost cleared away. It is now generally realized that three similar anomalies of the umbilicus may be evident at or shortly after birth: umbilical hernia, hernia into the cord, and omphalocele. In an umbilical hernia the defect is at the umbilical ring, but it is covered by skin and is unlikely to be recognized until the stump of the cord has dropped off and epithelization has occurred. In hernia into the cord the defect is also at the umbilical ring but the contents extruded from the abdomen are in the base of the cord. In omphalocele the defect is in the abdominal wall, not merely at the umbilical ring, and the contents of the sac, unlike the contents of the sac of an umbilical or funicular hernia, have never been in the peritoneal cavity. Whatever confusion remains would cease to exist, as Gross and Blodgett[8] emphasize, if some decisive term, such as omphalocele, were used for the anomaly in which the

defect is in the abdominal wall rather than in the umbilical ring. They prefer this term, but they also recognize the usefulness of such terms as exomphalos, eventration at the umbilicus, and amniotic hernia.

Etiologic Factors

The Charity Hospital records contribute nothing to the etiology of this condition, but it might be well to say a few words about the theories advanced to explain it. Although some of them are ingenious, most of them are completely lacking in universal application. At the present time, only two theories are worthy of consideration. The first, and most generally accepted, is that an omphalocele originates in a disproportion between the size of the fetal peritoneal cavity and the size of its contents. Between the sixth and tenth week of fetal life a large part of the intestinal tract of the child is displaced anteriorly into a recess in the base of the umbilical cord, which communicates with the yolk sac by means of the yolk stalk. Mall's explanation is entirely reasonable, that the enlargement of the fetal liver which occurs just previous to this period is so considerable that lack of space in the body cavity forces the intestines out into the expansible umbilical portion of the coelom. Subsequently, when growth of the lower abdominal cavity occurs at a normal rate, which is actually an accelerated rate, there is room for these organs to return to their normal habitat. If, however, growth is not sufficiently rapid, some abdominal viscera necessarily remain outside of the cavity and the child is born with an omphalocele.

The second of the two acceptable theories is advanced by Margulies,[9] who postulates that omphalocele results from failure of mesodermal structures in the transverse septum in the third week of fetal life. This theory explains the defect in the abdominal wall above the umbilicus, as well as the diaphragmatic and pericardial defects sometimes associated with it. In a case of omphalocele reported by Fox,[10] for instance, a diaphragmatic hernia was also present, and in another reported by Thompson[11] the heart and pericardium appeared outside of the chest wall. In 2 of my own 5 cases, as in other cases reported in the literature, exstrophy of the bladder was also present and the defect was below rather than above the umbilicus. Margulies' theory does not explain defects in this location.

Clinical Considerations

It is well to begin the clinical discussion of omphalocele by mentioning again the difference between it and funicular hernia or hernia of the cord. In the latter type of hernia there is a protrusion of fetal viscera through a normal or slightly enlarged open umbilical

ring into the cord at its base. Closure is easy and the only clinical significance of this anomaly, as has already been mentioned, is the possibility of obstructing the protruding intestine by ligating or clamping it along with the cord. An omphalocele, on the other hand, is associated with a major defect of the abdominal parietes. The sac, which is transparent or semitransparent, is composed of amnion and peritoneum, usually inextricably fused. The umbilical cord arises at or near the apex, and the umbilical vessels may be seen coursing on the under surface of the sac. Skin may extend upon the sac for a variable distance, but as a rule it ends close to the base, where it is continuous with the skin of the abdominal wall. The contained organs, as Kershner and his associates[4] say, are seen as if through a window pane, while Peikoff[12] vividly describes the sac and its contents as resembling a cellophane-wrapped basket of fruit. The diameter of the sac varies from 2 or 3 cm. to 12 to 15 cm. It may increase in size, sometimes considerably, when the child cries and the intestines are distended with air.

Delivery in all of the Charity Hospital cases was uncomplicated and as a rule was brief. A review of the literature, however, suggests that this is not always true. In Hemming's[3] case the omphalocele was a definite obstruction to delivery, and in Thompson's[11] case, in which a podalic version had to be done because of prolapse of a foot, the umbilical vessels had to be ligated and divided before delivery could be effected.

At birth the sac of the omphalocele is moist and pliable, but as it dries out with the passage of time, it tends to crack and rupture, and the risk of peritonitis increases as this process develops. The possibilities of contamination exist in every case. The sac was infected in 1 fatal case in the Charity Hospital series, and peritonitis was present in another fatal case when the child was first seen. Dense adhesions were present between the sac and the contents in 3 cases, none of which was fatal.

It is possible, and entirely conceivable, for the sac to be damaged at delivery. Sometimes, as in Maguire's[13] case, rupture of the sac may occur within the uterus; in his case fibrin was found on the exposed coils of intestine immediately after birth. More often it would seem that the sac has ruptured with the trauma of parturition. Hendtlass's[14] patient, for instance, was brought into the hospital partly delivered, with a loop of intestine about the shoulder. No sac was present in 4 of the 21 Charity Hospital cases, and the assumption is that it was ruptured during delivery, as the state of the intestines did not suggest long-standing exposure.

Almost all of the abdominal contents have at times been found in

an omphalocele, but the small intestine and portions of the liver are most frequently present. Occasionally all of the mobile intraabdominal contents are in the sac. The precise contents of the sac were not stated in 4 of the Charity Hospital series, all of which were fatal, but in 7 of the other cases, 5 of which were fatal, major portions of the liver were present in the sac in addition to the intestines, usually the ileum, cecum and colon. In one of these 5 cases the duodenum and pars pylorica were also present, and in another, also fatal, the stomach was present along with the small bowel and most of the colon.

The physiology of the displaced organs is surprisingly normal. They seem unaffected by alterations in environmental temperature, and even when the sac has ruptured at birth, they do not sustain irreparable damage if they are properly protected from contamination until they can be returned to the peritoneal cavity. As a rule strangulation does not occur, because of the wide opening of the sac. In Bryan's[15] case, however, in which almost all of the small intestine was in the sac, the bowel was so congested that it appeared nonviable on first inspection. A fibrous ring about the neck of the sac had to be cut before reduction could be accomplished, and a distinct compression ring was seen on the coils of intestine which had been caught in the opening.

Associated Anomalies

The congenital anomalies associated with omphalocele cover so wide a range that it is difficult to attempt to tabulate and classify them. In the 2 nonfatal and 5 fatal cases in this series in which other anomalies were present there were 4 instances of cardiac and 4 of vascular anomalies, practically all of which were serious; 5 anomalies of the genitalia; 5 of the urinary tract; 4 of the gastrointestinal tract; and 1 neurologic anomaly. Included in this list were such anomalies as the tetralogy of Fallot, persistence of the left vena cava, microphthalmia, absence of the large bowel, failure of development of the external genitalia and perineum, and renal agenesis. These 7 children also presented harelip, cleft palate, or both, abnormalities of bones, and 1 instance of the Klippel-Feil syndrome.

It is not very satisfactory to try to analyze the effect of these anomalies upon viability, but it is significant that the 2 children with associated defects who did not die immediately after operation returned within a period of months with serious feeding problems, from which both died. It seems fair to say that a single anomaly, even if serious, might be tolerated by the newborn child but that

multiple anomalies exert a cumulative deteriorating effect to which the child ultimately succumbs.

Therapy

The treatment of omphalocele is surgical, and urgently surgical. There is no hope that the skin will grow over the sac or that the contents may be returned to the peritoneal cavity by simple taxis. The clinical progress is invariably toward rupture of the sac as it dries out, infection ensues, and death is inevitable and not long delayed. There is therefore no warrant for attempts at conservative therapy. Failure to reduce the mass immediately after birth, before the intestines become distended with air and gas, is to lose a golden opportunity. Even if the child is cyanotic or if the abdomen is distended when he is first seen, it is best to operate with as little delay as possible. Oxygen may be administered and intestinal decompression carried out while preparations for operation are made.

There need be no fear that the newborn child, other things being equal, will not tolerate operation. He is likely to endure it remarkably well. Rogers,[16] who was obliged to perform what he termed a "manhandling" operation on his own patient, pointed out that this tolerance is to be expected. The operation, in his opinion, is simply an extension of birth trauma. Surgical obliteration of the extra-embryonic coelom can be regarded merely as an accelerated physiologic event brought about artificially by surgery, to which the trauma of birth has already adapted the child. Rogers' own operation was performed on a 20 minute-old child, whose whole liver was outside of the peritoneal cavity, and who evinced no shock, respiratory distress or other complication when it was replaced after a series of very trying maneuvers.

Anesthesia. My own preference is for open drop ether for anesthesia. I can see no point in the use of local analgesia, either alone or supplementally, nor can I see any point in operating without anesthesia. These children can be shocked by the pain of an operation performed without an anesthetic, and the lack of relaxation inevitably leads to more of what Rogers terms "manhandling." The newborn child endures anesthesia very well and it can be employed without fear of the consequences.

Technic. The objective of therapy in omphalocele is the closure of the defect in the abdominal wall, and when the mass is only a few centimeters in diameter, this is easily accomplished. When the sac is larger, however, especially when it exceeds 9 or 10 cm., closure is often extremely difficult and it takes nice judgment to determine whether to attempt to close the fascia and complete the operation

in one stage or to perform a two stage operation. There is, of course, more to the problem than returning the prodigal viscera to the peritoneal cavity and finding enough tissue to close the abdominal defect. One must also be certain that the return of the organs to their normal resting place does not raise the intra-abdominal pressure to a dangerous degree. When this happens, the consequences may be serious—intestinal obstruction, elevation of the diaphragm with diminution of the vital capacity, possibly failure of venous return to the right side of the heart, and circulatory collapse. These are the commonest causes of death after operation.

It was to obviate these dangers that Gross[17] first suggested that extensive skin flaps be undermined laterally and used to cover the viscera after excision of the sac, no attempt being made to close the fascia until a week or 10 days later, when the abdomen had become adjusted to its new contents. This technic, however, has two defects, as Gross himself pointed out. The first is that the repair is weak and it is always a question whether the suture line will hold and heal. The second is that the intestines are likely to become densely adherent to the broad, raw surface with which they are covered.

To overcome these dangers and disadvantages Gross[17] proposed another technic in 1948. By this new method the membrane of the omphalocele is left intact, the skin is cut free and widely undermined, and the cutaneous flaps thus formed are brought together anteriorly over the sac. The intestines are thus covered with a smooth membrane resembling normal peritoneum and the bowel itself has not been exposed to air or touched by instruments or gauze, while the pressure within the abdominal cavity has not been appreciably increased. Furthermore, the combination of intact membrane and superimposed skin is much stronger than the covering of skin used in the earlier operation. The bulging mass left on the abdomen is not aesthetic, it is true, but it is compatible with life, and secondary repair is possible, with closure of the muscles and fascia, some months later, when the cavity has increased in capacity.

This method was used in 2 cases in the Charity Hospital series, with survival in both instances. In the first case it was possible to cover the defect with the skin flaps after reduction of the mass but intra-abdominal pressure was so great that the child stopped breathing. Extensive relaxing incisions overcame this difficulty. The skin sloughed in the center of the wound, secondary closure was necessary, and healing was long delayed. The child left the hospital in reasonably good condition but returned three months later with a severe upper respiratory infection and feeding difficulties, to which he finally succumbed. Postmortem revealed a patent ductus arterio-

sus, a patent interauricular septum, and malrotation of the intestine with short mesenteric attachment.

In the second case in which the Gross technic was used, one of my own, infection also ensued, and four attempts were necessary before closure was effected. The abdomen had to be reopened, through another incision, four days after the last attempt, when manifestations of intestinal obstruction were followed by evisceration. Exploration showed a volvulus of the small bowel with obstruction of the terminal ileum, apparently the result of malrotation and a short mesenteric attachment. This is, incidentally, an anomaly whose possible association with omphalocele has not been sufficiently emphasized. The child in this case was finally discharged at the age of 4 months, with a large ventral hernia, which is still to be corrected.

It is very interesting to observe that a case in the Charity Hospital series, cared for by Dr. Edwin Mendelssohn in 1943, was managed by essentially the same principles as Gross advised in 1948. The omphalocele was the size of a grapefruit. The sac was buried and the stump of the cord was brought out through the lower angle of the wound, instead of being excised level with the skin. Death occurred from peritonitis, on the sixth postoperative day, following wound infection and evisceration. The course of events was easy to trace: This child was born 250 miles away from the hospital and there was considerable doubt about the precautions taken to avoid contamination. In cases such as this, when the sac is potentially infected and the natural peritoneal surface must be sacrificed in the interests of safety, perhaps it might be feasible to use some sterile plastic sheeting, such as polyethylene, to prevent crippling adhesions between the skin flaps and the bowel until definitive repair of the abdominal wall can be accomplished.

Several points of technic are important, whatever method is employed. The hypogastric arteries should be ligated as they enter the inferolateral parts of the abdominal wall and the umbilical vein ligated as it crosses the upper margin of the sac. The cord is amputated close to the sac when the Gross technic is used and is treated with a solution of half strength iodine, in an endeavor to prevent infection.

If the liver is so adherent to the sac that hemorrhage seems inevitable if attempts to separate it are made, it is best, even if the omphalocele is small, to replace it without removing the sac. I doubt the wisdom of attempting to reduce the size of a solid organ like the liver by squeezing it, as has been suggested, to force blood out of it.

It is recommended that the intestines be rotated as they are re-

placed, but anyone who has operated for this condition can appreciate Peikoff's[12] remark, that the surgeon is often fortunate to get them back at all. In his own case he jack-knifed the body as the peritoneum and fascia were closed. Three years after operation gastrointestinal studies showed that the intestines had rotated to an almost completely normal position except that the duodenum was curved upward and to the right.

Ley's[18] case indicates that if necessary, the normal position of even the liver may be disregarded. In this instance all the free contents of the abdomen except the spleen were in the sac and were returned to the cavity with some difficulty. A wide strip of the upper portion of the sac was left attached to the liver. Later x-ray studies showed the liver lying on the right side of the abdomen, with the right lobe uppermost. The superior surface was external, and it was presumed that the bile ducts and blood vessels lay anteriorly. The child had never been jaundiced and it was assumed that firm adhesions had probably formed and that future rotation of the organ, with twisting of the bile ducts and the vascular supply, was now very unlikely.

The edges of the sac should be freshened before closure, to encourage healing, but this should not be done until the possible excess of tissue has been clearly determined. If there is none to spare, this step must be omitted.

Mortality and Prognosis

In view of the presence of associated anomalies in many cases of omphalocele, which has already been discussed, it is impossible to make the flat statement that survival depends upon the time after birth at which surgery is done. Generally speaking, however, the longer the operation is deferred in a child free of anomalies incompatible with life, the poorer is the prognosis. O'Leary and Clymer,[1] in a review of the literature, found that there were 12 deaths in 56 cases when operation was done within 12 hours of birth, 21 per cent; 4 deaths in 9 cases when it was done within 24 hours, 44 per cent; and 8 deaths in 13 cases, 61 per cent, when it was done after this time.

In the Charity Hospital series there were 12 deaths in the 21 cases, a case fatality rate of slightly over 57 per cent. Operation was done at various times from an hour and a half to 45 hours after birth. With a single exception all the children who survived were operated on before the end of the seventh hour after delivery. In the remaining case operation was done at the end of 27 hours.

Benson and his associates[19] consider that the relation of the size

of the defect to the size and weight of the infant influences the prognosis, particularly if the child is premature. There were 2 premature children in the survivors in the Charity Hospital series, and, paradoxically, in the cases in which the exact size of the defect was stated the children with the largest defects were also among the survivors.

With 2 exceptions all of the 12 deaths in the series occurred in children who were in poor or borderline condition when they were operated on. The 2 children in apparently fair condition both died of peritonitis. The history of one of these cases (Mendelssohn's) has already been summarized. In the other case there was no evidence at all of a sac, though whether it was naturally absent, or had been destroyed at delivery, is not clear. At any rate, the source of the peritonitis, as in the case just mentioned, is obvious, since this child was also delivered outside of the hospital.

Ten children in this series were white and 11 Negro, the distribution being somewhat out of line with the hospital population, in which Negro admissions have predominated for more than a decade. There were 6 deaths among the 10 white children and the same number in the 11 Negro children. The racial factor obviously offers no explanation of the mortality in a condition of this sort. Moreover, even the children born outside of the hospital were brought for medical attention as rapidly as was possible.

The distribution of the deaths by years does, however, seem significant. In the 12 cases which occurred up to 1943 there were only 3 survivals, 1 in 1941 and 2 in 1942. There were no other cases until 1947. In the 9 cases which occurred after that time, there were 6 survivals. Two of the 3 deaths occurred in children who had major anomalies, the sum total of which was apparently incompatible with life. The third death, in 1949, was caused by aspiration pneumonia and might perhaps have been prevented by more careful nursing and an earlier resort to feeding by gavage. The child had a cleft palate and poor sucking reflex, which made for feeding difficulties.

I am quite sure in my own mind that the children who survived in these later years owe their lives to the more aggressive outlook upon omphalocele, the close cooperation between pediatric and surgical services, which brought them to the operating table as soon as possible after birth, and the greatly improved preoperative and postoperative management which characterizes all modern surgery. It is not an accident that there were no deaths from peritonitis, shock or circulatory failure in the last years of this study.

Omphalocele is a serious pediatric condition, with more serious

implications than the defect itself might suggest. The presence of associated anomalies may make the child's condition hopeless from the start. Nonetheless, the improvement which has occurred in the small series from the New Orleans Charity Hospital indicates, as does the larger series reported by Gross and his associates[17] from the Children's Hospital in Boston, that this is no longer a condition in which there is no hope. To those who may feel that it is, I recommend, as a wholesome tonic, a perusal of Dott's[7] article, written in 1932. He operated, an hour and a half after birth, "without enthusiasm," but the bonny baby who laughs out from the pages of the *Edinburgh Medical Journal* is living proof how richly rewarding vigorous efforts to save these unfortunate children can be.

Summary

A series of 21 omphaloceles from Charity Hospital of Louisiana at New Orleans, which occurred between 1938 and 1950 inclusive, has been analyzed from the clinical standpoint. The review was suggested by the author's personal experience in 5 cases which are included in the series and in 4 of which the immediate results were excellent. Associated anomalies sometimes make this condition one in which no surgery can be of any avail, but an aggressive attitude, close cooperation between pediatric and surgical services, and careful adjuvant management often produce excellent results in even apparently hopeless cases.

REFERENCES

1. O'Leary, C. M., and Clymer, C. E.: Umbilical hernia, Am. J. Surg. *52:*38 (April) 1941.
2. Jarcho, J.: Congenital umbilical hernia, Surg., Gynec. & Obst. *65:*593 (Nov.) 1937.
3. Hemming, G. R.: Case of exomphalos successfully treated by early operation, M. J. Australia *2:*490 (Oct. 5) 1946.
4. Kershner, D.; Zuckerman, I. C., and Kessler, L. N.: Omphalocele, J. Internat. Coll. Surgeons *11:*89 (Jan.-Feb.) 1948.
5. Ludwig, cited by Lindfors, in Jarcho.[2]
6. Kern, H. M.: New and simplified procedure for repairing omphalocele, Am. J. Surg. *77:*783 (June) 1949.
7. Dott, N. M.: Clinical record of case of exomphalos, illustrating embryonic type and its surgical treatment, Edinburgh M. J. *39:*105 (Aug.) 1932.
8. Gross, R. E., and Blodgett, J. B.: Omphalocele (umbilical eventration) in newly born, Surg., Gynec. & Obst. *71:*520 (Oct.) 1940.
9. Margulies, L.: Omphalocele (amniocele); its anatomy and etiology in relation to hernias of umbilicus and umbilical cord, Am. J. Obst. & Gynec. *49:*695 (May) 1945.
10. Fox, P. F.: Uncommon umbilical anomalies in children, Surg., Gynec. & Obst. *92:*95 (Jan.) 1951.
11. Thompson, D. J.: Obstructed labor with complicated presentation and exomphalos, Brit. M. J. *2:*45 (July 8) 1944.
12. Peikoff, S. S.: Exomphalos, Canad. M. A. J. *52:*600 (June) 1945.

13. Maguire, C. H.: Surgical management of omphalocele, Arch. Surg. *59:*484 (Sept.) 1949.
14. Hendtlass, R. F.: Case of extreme exomphalos, Brit. M. J. *1:*477 (April 17) 1943.
15. Bryan, T. B. L.: Case of exomphalos complicated by acute intestinal obstruction, Brit. M. J. *2:*818 (Dec. 23) 1944.
16. Rogers, L.: Exomphalos; radical cure in infant twenty minutes old, Brit. J. Surg. *29:*37 (July) 1941.
17. Gross, R. E.: New method for surgical treatment of large omphaloceles, Surgery *24:*277 (Aug.) 1948.
18. Ley, L.: Case of exomphalos successfully treated by operation, Brit. J. Surg. *30:*168 (Oct.) 1942.
19. Benson, C. D.; Penberthy, G. C., and Hill, E. J.: Hernia into umbilical cord and omphalocele (aminocele) in newborn, Arch. Surg. *58:*833 (June) 1949.

SERUM CHOLINESTERASE LEVELS IN SURGICAL PATIENTS

A Preliminary Report*

HOWARD E. SNYDER, M.D.
CECIL D. SNYDER, M.D.
LEITHA D. BUNCH, M.A.
Winfield, Kan.

IN 1939 Milhorat[1] reported his findings on the cholinesterase activity of the sera of 109 patients with various diseases. He found that in debilitated patients, the esterase level often was low and changed concomitantly with the clinical status of the patient. Antopol, *et al.*[2] reported serum cholinesterase activity in patients with jaundice or biliary tract disease to be depressed. Kunkel and Ward[3] studied the regeneration of plasma esterase in humans following the administration of DFP. The patients with liver cirrhosis showed a very slow formation of the enzyme. They concluded that the concentration of enzyme appearing in serum at any one time is a reflection of the rate of formation of the enzyme, by the liver. Vorhaus *et al.*[4,5,6] have reported studies indicating that serum cholinesterase activity is a better index of liver function than 10 other more commonly used tests. Alcalde[7] concludes that serum cholinesterase levels may be used to good advantage as a measure of liver function and as an aid in the differential diagnosis of jaundice. McCance[8] has found lowered serum cholinesterase activity in undernourished Germans with a return to normal as nutrition improved. Levine and Hoyt[9] have demonstrated that the level of serum cholinesterase varies directly with that of serum albumin in various pathological states, the one exception occurring in patients with albuminuria. The correlation does not exist when levels are normal or near normal.

This preliminary report concerns studies on 122 healthy controls and 150 surgical patients, but will deal chiefly with the data on 53 patients undergoing biliary tract surgery. In most instances, both preoperative and postoperative data are available and include a varying number of studies in addition to the determinations of serum cholinesterase activity. The number and timing of the studies made on each surgical case were dependent for the most part upon the general condition of the patient and not upon any set plan of study.

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*From the H. L. Snyder Memorial Research Foundation.

Presented during the Hollywood Assembly of The Southeastern Surgical Congress, Hollywood, Fla., Apr. 11-14, 1951.

The determination of serum cholinesterase activity was made by the continuous titration method of Hall and Lucas.[10] This test was found to be simple and highly reproducible. One unit of cholinesterase activity is the ml. of 0.01 N sodium hydroxide needed to neutralize the acetic acid liberated by 0.5 ml. serum from 25 mg. of acetylcholine chloride in 10 ml. of carbon dioxide free water in 10 minutes at pH 7.8 and temperature of 37° C.

Methods for potassium,[11] chlorides,[12] carbon dioxide content,[13] total proteins, albumin, globulin,[14] alkaline phosphatase,[15] bilirubin,[16] cephalin flocculation,[17] thymol turbidity,[18] cholesterol, cholesterol esters,[19] nonprotein nitrogen,[20] and hemoglobin[21] determinations may be found in the references given.

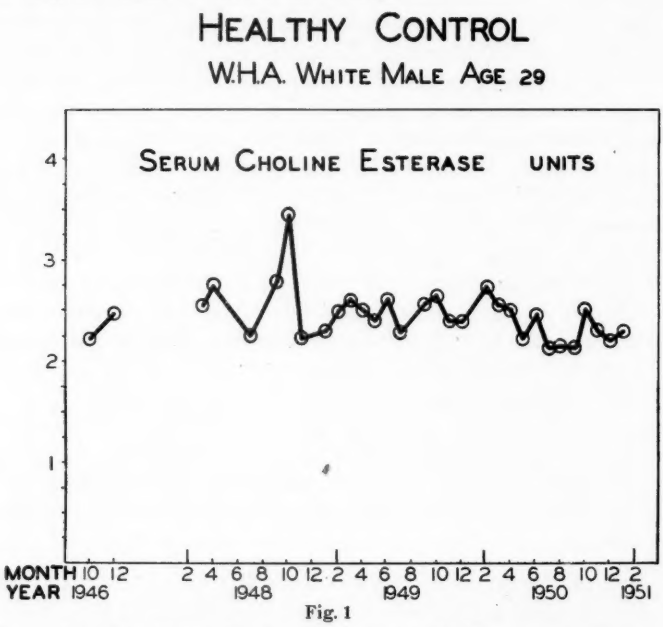

Fig. 1

From 2 to 31 determinations of serum cholinesterase were made on 122 healthy controls (table I). The mean value for the 61 males was 2.43 and for the 61 females 2.21 units. The mean value for the whole group was 2.32 units. While the difference in males and females is statistically significant, it is not of sufficient magnitude to be of any practical value in the consideration of one case.

In repeated determinations of cholinesterase in healthy controls, the relative constancy of the enzyme activity in each individual has been striking. Thirty-one determinations on one male control over a period of four years (fig. 2) showed a range in his cholinesterase

levels from 2.14 to 3.45 units with a mean of 2.46 units, standard deviation 0.26 and standard error 0.05.

Tables II to XIII present preoperative and postoperative laboratory data on individual cases of cholecystitis, cholelithiasis and choledocholithiasis. Cases are grouped according to surgery performed and diagnosis. Only one set of preoperative and one set of postoperative findings are shown. The postoperative data are of the day of the lowest serum cholinesterase activity recorded. Table XIV gives serial data on one case of choledocholithiasis. In studying these tables, one notes that the findings for cholinesterase and albumin seem consistent with the extent of disease and the surgery performed. They more closely paralleled the clinical estimate of

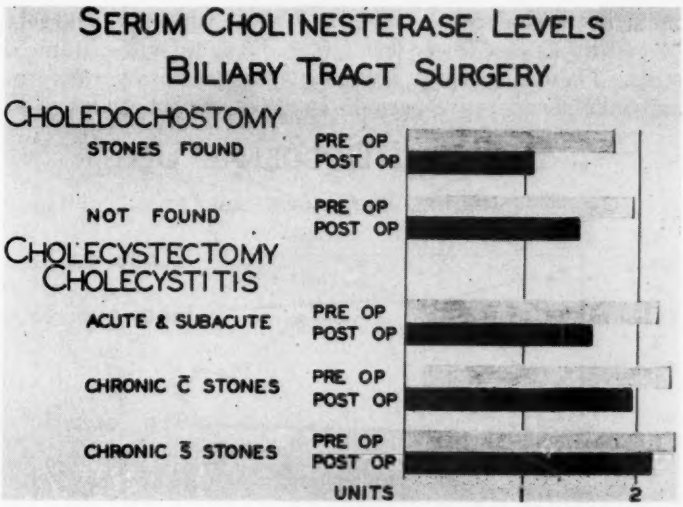

Fig. 2

the patient's course than any of the other laboratory tests. The percentage of cholesterol esters, while next in merit to cholinesterase and albumin levels, was not as predictable nor as valuable in the management of these surgical cases. Serum globulin tended to show an elevation with depression of cholinesterase and albumin levels, but this was not constant. The elevation of globulin was occasionally extreme and in these instances might have led to a rather false feeling of security if total protein levels had been accepted at face value. This is illustrated in the second case in table III in which the total proteins were 7.05 Gm. per cent, globulin 3.8 Gm. per cent, but albumin only 3.25 Gm. per cent and cholinesterase 1.05 units.

Findings with the cephalin flocculation test were not constant nor predictable. The thymol turbidity test proved of no value in the management of this group of cases. The alkaline phosphatase levels varied within wide limits tending to show the most elevation in those cases with common duct obstruction. The levels of serum bilirubin were, of course, valuable in the management of these cases but did not represent an index of liver function.

Comparing serum cholinesterase levels in the group undergoing choledochostomy with those in which only cholecystectomy was performed, one finds significantly lower levels in the former (tables XV, XVI, and fig. 2). The cases having choledochostomy in which common duct stones were found, had a lower cholinesterase level than did those in which common duct exploration did not reveal stones. In the cholecystectomy group, those with acute and subacute cholecystitis showed lower levels than did those with chronic cholecystitis. These differences are greater in the postoperative period than in the preoperative period. In all of the subgroups of biliary

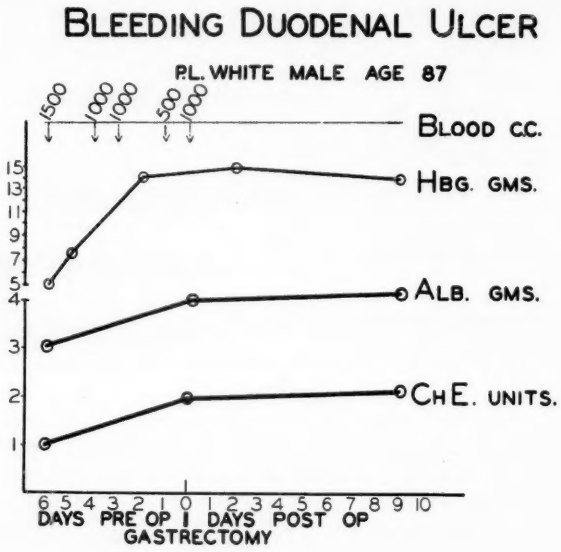

Fig. 3

tract surgery, the mean postoperative cholinesterase levels were lower than the preoperative levels. While the albumin levels tended to parallel cholinesterase levels, they did not seem to indicate as accurately the extent of surgery and disease.

Patients with low hemoglobin levels showed varying degrees of depression of serum cholinesterase and albumin levels. One patient

(41192) with an acute hemorrhage from a duodenal ulcer is illustrated (fig. 3). In the six days prior to operation, he was given 4000 ml. of blood, the serum cholinesterase rose from 1.05 units to 2.04 units and the hemoglobin from 5.1 to 13.95 Gm. per cent. Table XVII presents preoperative data on patients having gastrectomy for peptic ulcer. Included are other cases showing preoperative elevation of cholinesterase activity following blood transfusion. Case 42179 showed an increase in cholinesterase activity from 1.07 to 1.5 units with the transfusion of 1500 ml. of blood in a two day period. On the other hand, one case (6707) showed a cholinesterase level of only 1.19 units after transfusion of 3500 ml. of blood although hemoglobin had risen from 8.5 to 15 Gm. per cent.

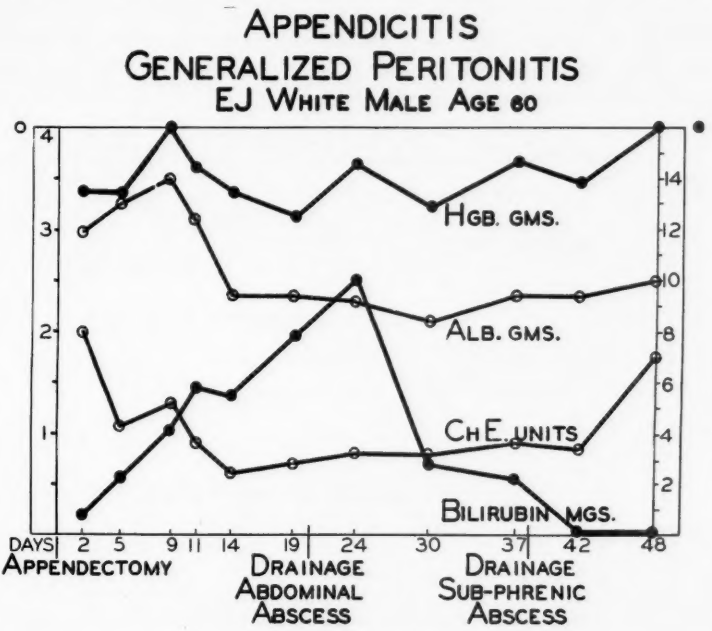

Fig. 4

In this case, bleeding had been taking place over a period of at least 30 days prior to admission. From these data, one might postulate that disturbance of liver function by hemorrhage varied with the degree and duration of the anemia produced.

There was a striking difference in the postoperative cholinesterase levels of those having gastrectomy for ulcer and those having choledochostomy. The levels of the gastrectomy group (table XVIII) were much higher. The gastrectomies rarely showed any great depression of cholinesterase activity even with a complicated post-

operative course. On the other hand, a definitely depressed level of serum cholinesterase activity was the rule in cases having choledochostomy and cases having cholecystectomy for acute or subacute cholecystitis.

The influence of infection within the peritoneal cavity upon serum cholinesterase activity is illustrated (fig. 4). This 60 year old male with severe generalized peritonitis from appendicitis showed a low serum cholinesterase level of 0.615 units, 14 days after an appendectomy. The degree of toxic hepatitis produced is demonstrated by a serum bilirubin level of 9.72 mg. per cent on the twenty-fourth postoperative day. Clinical improvement in this case started after the surgical drainage of an abdominal abscess on the twentieth postoperative day and pronounced improvement followed the drainage of a subphrenic abscess on the thirty-eighth postoperative day. Ten days following the latter procedure, his serum cholinesterase activity had risen to 1.75 units.

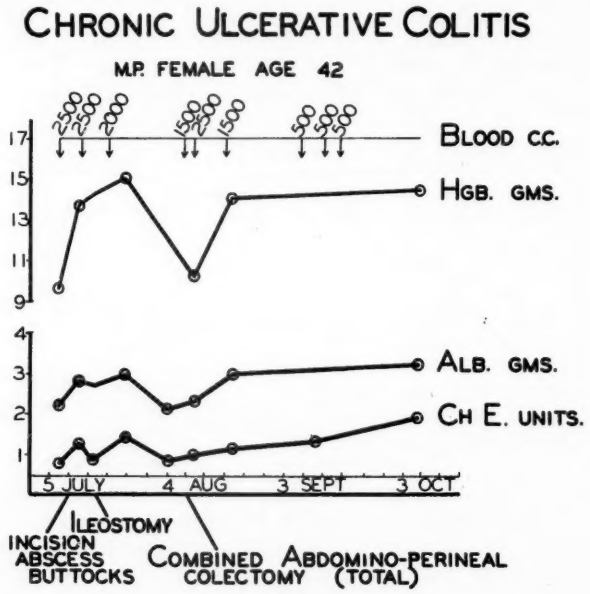

Fig. 5

The effect of chronic infection, loss of blood and loss of protein through exudation is illustrated (fig. 5) which gives data on a case of chronic ulcerative colitis of 15 years standing. In the 13 days prior to doing an ileostomy under local anesthesia, 5000 ml. of blood were transfused. In the next 28 days, 3500 ml. additional blood were transfused but continuing loss of blood and protein from

the bowel was so great that the general condition of the patient was not improving. It was elected at this time to proceed with the total colectomy. On the day of this operation, 2500 ml. of blood were transfused. While her postoperative course was not alarming at any time, 3000 ml. of blood were transfused in the 40 days following surgery to maintain a satisfactory hemoglobin level. In the last 20 days of her hospital stay, she maintained a satisfactory hemoglobin level and showed increases in both serum cholinesterase and albumin levels, without transfusion.

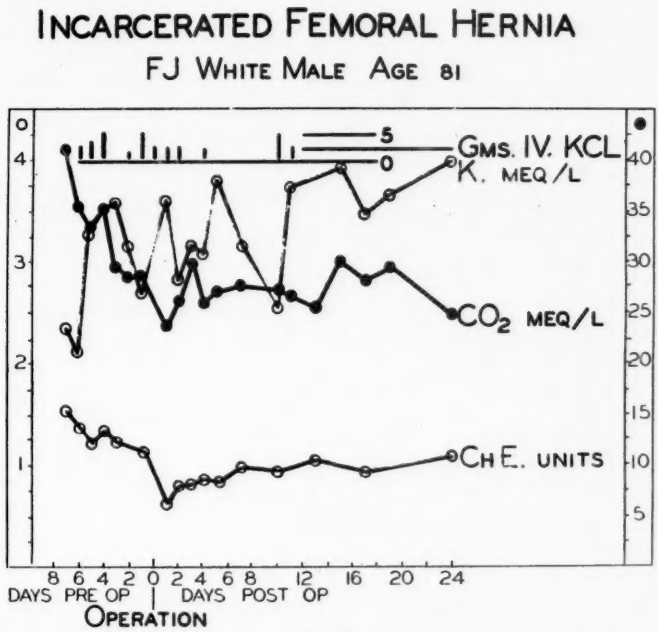

Fig. 6

Figure 6 shows the effect on cholinesterase activity of prolonged intestinal obstruction due to incarcerated hernia. This obstruction had been present for five days prior to hospital admission. Eight more days were required to correct the extreme alkalosis before surgery could be attempted for relief of the obstruction. The cholinesterase activity fell to a low of 0.63 units 24 hours after operation, then very gradually increased in the postoperative period.

Those cases with benign prostatic obstruction tended to show low cholinesterase levels. This is illustrated (fig. 7), depicting a case in which the admission nonprotein nitrogen level was 54.5 mg. per cent, hemoglobin 9.4 Gm. per cent, albumin 3.85 Gm. per cent,

and cholinesterase 1.17 units. The cholinesterase level rose with transfusion and catheter drainage to 1.64 units as the nonprotein nitrogen fell to 49.6 mg. per cent. With postoperative anemia, the cholinesterase level fell to 1.18 units. In five days, it rose to 1.36 units while the hemoglobin had increased from 9.7 to 15.7 Gm. per cent.

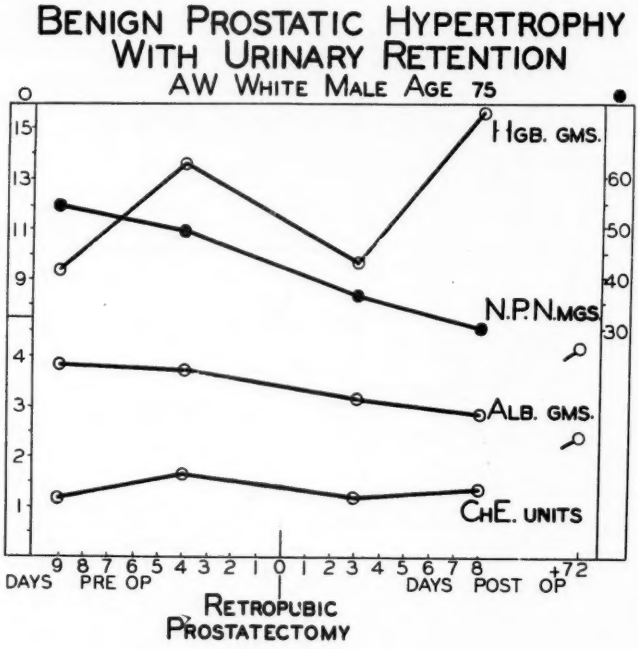

Fig. 7

The complete data on the surgical cases, other than those with biliary tract disease, will not be reported at this time. Preliminary study seems to indicate that cholinesterase activity is not depressed to any degree unless there is some factor present leading to impairment of liver function. Those having hysterectomy for fibroids tend to have near normal levels unless there has been hemorrhage. In malignancy, the esterase activity tends to be low but only if there is actual liver involvement, blood loss or other pathology which might impair liver function.

Prior to statistical study, we had the impression that serum cholinesterase levels tended to parallel albumin levels, but that the cholinesterase level was the more sensitive and accurate of the two measurements. However, analysis of the data on 122 controls and 120 surgical cases provided some very interesting findings (table

TABLE I

Serum Cholinesterase Levels in Healthy Controls

	Cases	Determinations*	Mean	Standard Deviation	Standard Error
Male	61	438	2.43	0.32	±0.04
Female	61	310	2.21	0.32	±0.04
Total	122	748	2.32	0.35	±0.03

*Mean of all determinations on each case used in computing mean for each group.

TABLE II

Choledochostomy-Choledocholithiasis

Preoperative Laboratory Findings

Case No.	Age	Sex	Preop. Day	ChE Units	Alb. Gms. %	Glob. Gms. %	Chol. Esters %	Ceph. Floc. 48 Hrs.	Thymol Turb.	Alk. Ptase. Units	Bili-rubin Mg. %	Hgb. Gms. %
40447	55	F	0	0.93			83.00	1+	0.00	25.0	2.50	11.20
30966	73	F	4	1.19	3.85	2.65	71.60	neg.			0.72	14.20
20772	72	M	9	1.61			52.00	4+	0.80	1.0	2.10	13.55
15376	68	F	5	2.12	4.40	2.40	63.80	neg.		43.0	2.76	12.20
31785	38	F	1	1.61	3.85	2.25	77.50	neg.		13.5	4.34	13.20
33605	45	F	2	2.28	3.90	1.90	70.00	1+	0.57	0.3	0.40	12.80
616	63	M	0	2.68	3.90	1.80	63.50		0.10		8.20	11.05
14644	82	F	6	1.87	4.35	2.15	75.00	2+	0.30	21.5	0.72	13.00
43336	60	M	7	1.30	4.50	1.75	58.50	2+		100.0	5.10	13.30
Mean				1.73	4.11		68.32					
Standard deviation of mean				0.55	0.22		9.23					
Standard error of mean				0.18	0.09		3.08					

TABLE III

Choledochostomy-Choledocholithiasis

Postoperative Laboratory Findings on Day of Lowest Serum Cholinesterase Activity

Case No.	Age	Sex	Postop-Day	ChE Units	Alb. Gms. %	Glob. Gms. %	Chol. Esters %	Ceph. Floc. 48 Hrs.	Thymol Turb.	Alk. Ptase. Units	Bili-rubin Mg. %	Hgb. Gms. %
40447	55	F	7	1.19	3.25	2.45					1.00	
30966	73	F	17	1.05	3.25	3.80	79.00	3+		12.00	2.34	15.40
20772	72	M	12	1.11	3.40	3.40	77.00		1.10	1.50		
15376	68	F	23	1.30	3.70	2.20	67.00	neg.		50.00	0.81	10.90
31785	38	F	8	0.57	2.35	2.25	43.70	1+		7.00	4.36	13.80
33605	45	F	2	1.36	2.70	2.00	68.50		0.50	0.50	1.45	
616	63	M	13	0.81	2.60	2.05	44.00		1.45		4.75	11.05
4644	82	F	5	0.80	3.15	1.60	68.00	neg.		9.00	0.81	10.60
43336	60	M	8	1.23	3.65	1.95	77.00	1+		37.00	0.63	12.60
Mean				1.05	3.12		65.20					
Standard deviation of mean				0.23	0.41		13.02					
Standard error of mean				0.08	0.14		4.60					

TABLE IV

Choledochostomy-No Stones Found

Preoperative Laboratory Findings

Case No.	Age	Sex	Preop. Day	ChE Units	Alb. Gms. %	Glob. Gms. %	Chol. Esters %	Ceph. Floc. 48 Hrs.	Thymol Turb.	Alk. Ptase. Units	Bili-rubin Mg. %	Hgb. Gms. %
9556	44	F	1	1.59	3.35	2.05	66.7	3+		9.0	0.72	12.8
23647	62	F	1	1.94	4.10	1.75	71.7	1+	0.3	13.0	1.35	12.0
13561	63	F	3	1.26	3.70	2.15	50.0	±	4.8	14.4	18.90	11.5
30311	52	M	2	1.92	3.45	2.05	73.0	1+	1.1	6.0	2.40	

38145	79	F	5	2.34	2.85	2.87					0.43	
2436	74	F	3	2.15	3.10	2.70						14.2
170	49	F	1	1.44	3.70	1.70	80.00	1+		2.0	0.72	12.0
Mean				1.81	3.46		68.28					
Standard deviation of mean				0.34	0.42		10.18					
Standard error of mean				0.13	0.16		4.55					

TABLE V

Choledochostomy-No Stones Found

Postoperative Laboratory Findings on Day of Lowest Serum Cholinesterase Activity

Case No.	Age	Sex	Postop. Day	ChE Units	Alb. Gms. %	Glob. Gms. %	Chol. Esters %	Ceph. Floc. 48 Hrs.	Thymol Turb.	Alk. Ptase. Units	Bili-rubin Mg. %	Hgb. Gms. %
13561	63	F	5	1.43	3.40	2.15	47.2	±	0.90	5.0	11.20	14.80
30311	52	M	13	1.60			51.0	+	0.45	6.0	0.92	
38145	79	F	18	1.36	3.60	2.70	67.0		0.50			11.65
2436	74	F	20	1.57	3.00	3.00	70.4	2+	0.60	7.0	2.60	
170	49	F	9	1.11	2.95	1.95				7.0	0.10	12.50
Mean				1.41	3.25		58.90					
Standard deviation of mean				0.21	0.26		9.96					
Standard error of mean				0.09	0.13		4.98					

TABLE VI

Cholecystectomy-Acute and Subacute Cholecystitis

Preoperative Laboratory Findings

Case No.	Age	Sex	Preop. Day	ChE Units	Alb. Gms. %	Glob. Gms. %	Chol. Esters %	Ceph. Floc. 48 Hrs.	Thymol Turb.	Alk. Ptase. Units	Bili-rubin Mg. %	Hgb. Gms. %
33034	57	F	11	2.48	4.35	2.05	81.00	2+	0.20	2.90	0.44	
40051	58	F	1	2.68	3.90	1.90	87.00	neg.	1.30	3.00	1.38	12.80

40035	69	F	Op.	2.64	3.05	3.00	56.00	neg.	1.10	5.00	1.17	13.20
33380	53	F	2	2.33	3.70	1.90	73.00		0.50			13.60
18350	65	M	1	1.75	3.65	1.75	64.50	neg.	0.20	4.00	1.25	13.30
40359	43	F	7	1.45	3.95	2.30	74.50	neg.		6.00	0.26	11.30
31645	60	M	5	2.43	4.20			1+				14.35
20397	64	F	7	1.72	3.40	2.70	73.00	neg.		3.20	0.09	12.50
6118	53	F	5	2.47	4.50	1.40	77.00	neg.	0.54	3.50	1.20	12.80
743	51	F	Op.	1.74	4.30	2.65	81.00	neg.		8.00	0.63	13.30
Mean				2.16	3.90		74.10					
Standard deviation of mean				0.47	0.43		8.86					
Standard error of mean				0.15	0.14		2.95					

TABLE VII

Cholecystectomy-Acute and Subacute Cholecystitis

Postoperative Laboratory Findings on Day of Lowest Serum Cholinesterase Activity

Case No.	Age	Sex	Postop. Day	ChE Units	Alb. Gms. %	Glob. Gms. %	Chol. Esters %	Ceph. Floc. 48 Hrs.	Thymol Turb.	Alk. Ptase. Units	Bili-rubin Mg. %	Hgb. Gms. %
40051	58	F	2	1.89								11.20
40035	64	F	9	1.13	2.50	2.50	64.00	neg.	0.70	28.00	0.80	11.00
33380	53	F	1	1.95	3.20	2.20	73.00		0.30			
18350	65	M	2	0.96	2.95	1.85	65.50	neg.	0	5.00	0.54	12.20
31645	60	M	1	1.72	2.60			2+			1.35	
20397	64	F	2	1.54	3.90	2.35	77.00			6.00		13.00
743	51	F	3	1.35	3.25	2.45	89.00	neg.		8.50	0.10	
Mean				1.51	3.06		73.70					
Standard deviation of mean				0.33	0.46		8.95					
Standard error of mean				0.12	0.19		4.01					

TABLE VIII

Cholecystectomy-Chronic Cholecystitis and Cholelithiasis

Preoperative Laboratory Findings

Case No.	Age	Sex	Preop. Day	ChE Units	Alb. Gms. %	Glob. Gms. %	Chol. Esters %	Ceph. Floc. 48 Hrs.	Thymol Turb.	Alk. Ptase. Units	Bili-rubin Mg. %	Hgb. Gms. %
2089	47	F	1	2.21	4.10	1.70	73.00	neg.	0.00	3.00	0.20	14.75
38970	50	M	2	2.68	3.60	2.00	79.00		0.40	3.20	0.40	12.60
6411	26	F	4	2.36	4.54	2.06	48.00	1+	0.60	4.50	0.40	14.35
13580	69	F	2	2.32			73.00		0.30	7.50		14.35
2626	67	F	5	2.35	3.55	2.10	75.00	neg.	0.10	5.00	neg.	13.05
33309	43	M	1	2.04	3.70	1.80	73.00	2+	0.00	2.00	0.06	15.20
258	54	M	1	2.50	3.40	1.75	88.00	1+	0.55	5.00	0.20	14.75
7907	72	F	1	2.14	3.40	2.30	68.00				0.89	13.95
6501	47	F	1	1.83	3.90	1.40	71.00		0.60	4.60	1.09	13.55
28754	44	F	2	1.91			78.50		1.20	1.10	0.10	13.15
1966	38	M	5	2.30	3.60	2.00	71.00	neg.	0.25	3.10	0.10	13.05
Mean				2.24	3.75		72.50					
Standard deviation of mean				0.24	0.39		24.91					
Standard error of mean				0.07	0.13		7.51					

TABLE IX

Cholecystectomy-Chronic Cholecystitis and Cholelithiasis

Postoperative Laboratory Findings on Day of Lowest Serum Cholinesterase Activity

Case No.	Age	Sex	Postop. Day	ChE Units	Alb. Gms. %	Glob. Gms. %	Chol. Esters %	Ceph. Floc. 48 Hrs.	Thymol Turb.	Alk. Ptase. Units	Bili-rubin Mg. %	Hgb. Gms. %
2089	47	F	4	2.00	4.20	1.60	86.70		0.35	7.50	0.60	
6411	26	F	1	2.28	4.54	1.86	79.00	1+	0.30	1.75	0.20	

3937	31	F	1	2.13	4.10		73.00		0	1.50	0.20	
33309	43	M	2	1.85	3.45	2.25	64.00		0		0.40	
258	54	M	8	2.22	3.80	2.00	71.00	1+	1.20	1.20	0.15	
28754	44	F	8	1.60	3.20	2.00	81.00		1.30	1.60	0.30	
1966	38	M	2	1.57	3.60	2.00	70.00		0	1.40		12.6
Mean				1.95	3.84		74.95					
Standard deviation of mean				0.27	0.44		14.33					
Standard error of mean				0.10	0.17		2.65					

TABLE X

Cholecystectomy-Chronic Cholecystitis-No Stones Found

Preoperative Laboratory Findings

Case No.	Age	Sex	Preop. Day	ChE Units	Alb. Gms. %	Glob. Gms. %	Chol. Esters %	Ceph. Floc. 48 Hrs.	Thymol Turb.	Alk. Ptase. Units	Bili-rubin Mg. %	Hgb. Gms. %
23188	43	F	2	1.95	4.13	2.47	85.0	trace	0.60		0.20	14.35
37968	42	F	Op.	3.02	4.00	2.05	79.0		0.80		1.00	12.15
5060	36	F	Op.	2.40	4.54	1.96	56.5		0.26		0.60	13.15
8597	41	F	2	1.60	3.90	2.10	73.0	neg.	0.00	1.1	0.80	13.95
19788	62	F	Op.	1.70	3.95	2.30	73.5			2.7	0.25	12.15
30700	45	F	1	2.57	4.30	1.95	73.5		2.10	1.0	0.80	12.15
14911	60	F	1	2.82	3.30	1.55	72.6	1+	0.30	2.0	0.30	12.80
5778	41	F	1	2.90	4.10	2.85	78.0	+ —	70.10	9.2	0.79	12.60
41284	56	M	7	2.40	4.60	1.80	72.6	neg.	0.30		0.40	12.20
42184	45	M	14	1.90	3.95	2.70	75.5	4+			0.50	11.70
Mean				2.32	4.02		73.87					
Standard deviation of mean				0.51	0.76		23.45					
Standard error of mean				0.16	0.24		7.41					

TABLE XI

Cholecystectomy-Chronic Cholecystitis-No Stones Found

Postoperative Laboratory Findings on Day of Lowest Serum Cholinesterase Activity

Case No.	Age	Sex	Postop. Day	ChE Units	Alb. Gms. %	Glob. Gms. %	Chol. Esters %	Ceph. Floc. 48 Hrs.	Thymol Turb.	Alk. Ptase. Units	Bili-rubin Mg. %	Hgb. Gms. %
23188	43	F	1	1.99	3.40	2.10	72.5		0.6		0.25	
8597	41	F	10	1.66	3.95	1.95		1+	0	1.2	0.10	11.7
30700	45	F	3	2.26	4.30	2.50	63.0		1.7	3.5	1.40	
14911	60	F	8	2.12	2.90	2.00	67.0	2+	1.2	2.7	0.20	
5778	41	F	1	3.01	3.50	3.45	81.0	neg.	0	2.3	1.03	13.2
38766	34	M	4	1.69				neg.	0.2			14.0
Mean				2.12	3.61		70.87					
Standard deviation of mean				0.50	0.48		6.80					
Standard error of mean				0.20	0.21		3.40					

TABLE XII

Cholecystectomy-Chronic Cholecystitis Complicated by Other Disease

Preoperative Laboratory Findings

Case No.	Age	Sex	Preop. Day	ChE Units	Alb. Gms. %	Glob. Gms. %	Chol. Esters %	Ceph. Floc. 48 Hrs.	Thymol Turb.	Alk. Ptase. Units	Hgb. Gms. %
921	72	F	2		3.15	2.05					13.15
39484	76	M	24	1.70	3.85	2.00	69	neg.	0.15	2.7	
38705	78	M	4	1.34	3.35	2.65	71	4+	1.65	2.0	12.80
Mean				1.52	3.42		70				
Standard deviation of mean				0.18	0.54		1				
Standard error of mean				0.13	0.31		0.71				

TABLE XIII

Cholecystectomy-Chronic Cholecystitis Complicated by Other Disease

Postoperative Laboratory Findings on Day of Lowest Serum Cholinesterase Activity

Case No.	Age	Sex	Postop. Day	ChE Units	Alb. Gms. %	Glob. Gms. %	Chol. Esters %	Ceph. Floc. 48 Hrs.	Thymol Turb.	Alk. Ptase. Units	Bili-rubin Mg. %	Hgb. Gms. %
921	72	F	1	1.39								16.00
39484	76	M	6	0.88	2.45	2.35	75.00	neg.	0.70	3.30	0.60	
41522	69	F	13	1.25								
38705	78	M	26	0.90	2.75	1.95	34.00	3+	0.98	2.60	1.10	
Mean				1.10	2.60		55.00					
Standard deviation of mean				0.24	0.15		20.50					
Standard error of mean				0.12	0.11		14.50					

TABLE XIV

Choledochostomy-Cholecystectomy

Chronic Cholecystitis, Cholelithiasis, Choledocholithiasis 14644-White Female-Age 82

Time Interval Days	Cholin-esterase Units	Alb. Gms. %	Glob. Gms. %	Chol. Esters %	Ceph. Floc. 48 hrs.	Alk. Ptase.	Bili-rubin Mg. %	Hemo-globin Mg. %
—6	1.55	4.10	2.40	87.6	neg.	21.5	1.2	13.2
Surgery								
+5	0.80	3.15	1.60	68.0	neg.	9.0	0.81	10.6
+17	0.95	2.35	2.85	74.0	2+	13.5	1.17	13.0
+25	1.11	2.00	3.70	75.0	neg.	7.0	0.81	13.8
+33	1.35	2.85	2.45		3+	6.0	0.09	
+40	1.50	3.15	2.25		3+			14.3
+81	1.91	3.45	3.05	83.0	2+	7.5	0.36	13.0

TABLE XV

Biliary Tract Surgery

Preoperative Mean Levels of Serum	Cholinesterase Units	Cases	Albumin Gms. %	Cases	Cholesterol Esters %	Cases
Choledochostomy						
Stones found	1.73±0.18	(9)	4.11±0.9	(7)	68.32±3.08	(9)
No stones	1.81±0.13	(7)	3.46±0.16	(7)	68.28±4.55	(5)
Cholecystectomy						
Cholecystitis						
Acute and subacute	2.16±0.15	(10)	3.9 ±0.14	(7)	74.1 ±2.95	(9)
Chronic with stones	2.24±0.07	(11)	3.75±0.13	(11)	72.5 ±7.5	(11)
Chronic without stones	2.32±0.16	(10)	4.02±0.24	(10)	73.87±7.4	(10)
Chronic with complicating disease	1.52±0.13	(2)	3.42±0.11	(3)	70.0 ±0.7	(2)

TABLE XVI

Biliary Tract Surgery Postoperative Mean Levels

	Cholinesterase Units	Cases	Albumin Gms. %	Cases	Cholesterol Esters %	Cases
Cholecystectomy and Choledochostomy						
Stones found	1.05±0.08	(9)	3.05±0.15	(9)	65.2±4.6	(8)
No stones	1.41±0.9	(5)	3.33±0.17	(5)	58.9±5.0	(4)
Cholecystectomy						
Cholecystitis						
Acute and subacute	1.51±0.12	(10)	3.06±0.19	(7)	73.7±4.01	(4)
Chronic with stones	1.95±0.1	(7)	3.84±0.17	(7)	74.9±2.64	(11)
Chronic without stones	2.12±0.21	(6)	3.61±0.21	(5)	70.8±3.4	(4)
Chronic with complicating disease	1.10±0.12	(3)	2.6 ±0.11	(2)	54.5±14.5	(2)

TABLE XVII

Gastrectomy for Peptic Ulcer Preoperative Laboratory Findings

Case No.	Day Pre-operative	Cholinesterase Units	Albumin Gms. %	Cholesterol Esters %	Hemoglobin Gms. %	Remarks
42179	2	1.07			9.15	15000 cc. blood given
	0	1.50			9.15	between determinations
17822	4	2.50	4.10	81.00	14.75	
41192	6	1.05	3.05	78.00	5.10	4000 cc. blood given
	0	2.04	3.90	78.00	13.95	preoperatively
35505	10	1.93	3.70	73.00	14.50	5000 cc. blood given before this study
8266	2		3.90		13.15	
23267	1	2.63	3.50	78.00		
28336	26	1.68				1450 cc. blood given before this study
6707	2	1.19	3.85	78.00	13.30	3500 cc. blood given before this study

The following case numbers have no remarks:

Case No.	Cholesterol Esters %	Hemoglobin Gms. %
17822		14.75
8266		13.15
23267	78.00	

TABLE XVIII

Gastrectomy for Peptic Ulcer

Postoperative Laboratory Findings on Day of Lowest Serum Cholinesterase Activity

Case No.	Day Post-operative	Cholinesterase Units	Albumin Gms. %	Cholesterol Esters %	Hemoglobin Gms. %
42179	2	1.54	3.85		11.70
17822	2	2.22	4.20	69.00	16.00
41192	9	2.07	4.05	76.00	14.00
8266	15	1.84		65.00	13.95
23267	13	2.04	3.60	75.00	14.75
28336	1	1.84	3.40		13.30
6707	2	1.81	3.85	86.00	14.70
Mean		1.91	3.83	74.20	
Standard deviation of mean		0.19	0.27	7.14	
Standard error of mean		0.07	0.10	3.15	

TABLE XIX

Coefficient of Correlation Between Cholinesterase and Albumin

Groups	Determinations	Cases	Coeff. Corr.* ± S.E.	Correlation
Normals	748	122	+0.02±0.09	None
Biliary tract surgery	146	44	+0.4 ±0.7	Highly significant
Surgical carcinoma	76	32	+0.4 ±0.1	Highly significant
All surgical cases except carcinoma and biliary tract	133	44	+0.53±0.06	Highly significant
All cholinesterase determinations above 2 in surgical cases	72	48	+0.06±0.12	None
All cholinesterase determinations below 2 in surgical cases	283	99	+0.5 ±0.04	Highly significant

*Pearsonian coefficient of correlation.

XIX). The coefficient of correlation between cholinesterase and albumin in 748 determinations on 122 normal controls was $+0.02 \pm 0.09$, indicating no correlation in the controls.

Analysis of 146 determinations on 44 cases having biliary tract surgery showed a highly significant correlation between the serum albumin and serum cholinesterase levels. The same was true of 76 determinations on 32 cases of surgical carcinoma, and 133 determinations on 44 additional surgical cases. However, when all of the determinations on the surgical cases were divided into those having a serum cholinesterase level of 2 units or above, and those with levels below 2 units, a highly significant correlation was found only in the latter group. These findings are in agreement with those of Levine and Hoyt.[9]

Summary

1. The mean serum cholinesterase level in 122 healthy controls was 2.32 units ± 0.032.

2. There was a highly significant difference between the mean levels in male and female.

3. In the surgical cases studied, the levels of serum cholinesterase and serum albumin were better indices of liver function and the general condition of the patient than the other laboratory tests of liver function employed (total serum proteins, serum globulin, percentage of cholesterol esters, cephalin flocculation, thymol turbidity, alkaline phosphatase, and serum bilirubin).

4. In patients undergoing surgery upon the biliary tract, preoperative and postoperative serum cholinesterase levels varied with the extent of disease and the surgery performed. Those having choledochostomy for common duct stones showed a significantly lower postoperative serum cholinesterase level than did those having choledochostomy, when no common duct stones were found. The levels in those having cholecystectomy for acute or subacute cholecystitis were significantly lower than those having cholecystectomy for chronic cholecystitis.

5. Postoperative serum cholinesterase levels in those having subtotal gastrectomy were significantly higher than the levels in those having choledochostomy and those having cholecystectomy for acute and subacute cholecystitis.

6. In those cases with acute severe bleeding, cholinesterase activity fell to a low level but returned rapidly to a near normal level after prompt and adequate replacement of blood.

7. In those with prolonged or chronic bleeding with moderate or

severe anemia, low cholinesterase levels improved slowly after adequate replacement of blood and elimination of the source of bleeding.

8. Data are presented graphically on individual patients showing depressed cholinesterase levels in chronic ulcerative colitis, appendicitis with peritonitis, prolonged intestinal obstruction from incarcerated hernia, and prostatic obstruction.

9. In 120 surgical patients, there was a highly significant correlation between the levels of serum cholinesterase and serum albumin.

Conclusions

Serum cholinesterase activity and albumin levels constitute excellent indices of liver function and of the general condition of surgical patients. The determination of cholinesterase is a more specific measurement of a single protein of liver origin than is the determination of serum albumin by the commonly available chemical procedures. The determination of cholinesterase has been found a simple and highly reproducible test which has proven useful in the management of major surgical cases.

References

1. Milhorat, A. T.: Cholinesterase activity of blood serum in disease, J. Clin. Investigation *17:*649 (Sept.) 1938.
2. Antopol, W.; Schifrin, A., and Tuchman, L.: Decreased cholinesterase activity of serum in jaundice and in biliary disease, Proc. Soc. Exper. Biol. & Med. *38:*363 (April) 1938.
3. Kunkel, H. G., and Ward, S. M.: Plasma esterase activity in patients with liver disease and nephrotic syndrome, J. Exper. Med. *86:*325 (Oct.) 1947.
4. Vorhaus, L. J., and others: Serial serum cholinesterase determinations in liver disease, J. Lab. & Clin. Med. *33:*1475, 1948.
5. Vorhaus, L. J.; Scudamore, H. H., and Kark, R. M.: Measurement of serum cholinesterase activity in study of diseases of liver and biliary system, Gastroenterology *15:*304, 1950.
6. Vorhaus, L. J.; Scudamore, H. H., and Kark, R. M.: Measurement of serum cholinesterase activity, a useful test in management of acute hepatitis, Am. J. M. Sc. *221:*140 (Feb.) 1951.
7. Alcalade, J. M. O.: Serum cholinesterase determination in differential diagnosis of jaundice, J. Lab. & Clin. Med. *36:*391 (Sept.) 1950.
8. McCance, R. A.: Serum cholinesterases in undernutrition, Proc. Roy. Soc. Med. *43:*272, 1950.
9. Levine, M. G., and Hoyt, R. E.: Relationship between human serum cholinesterase and serum albumin, Science *111:*286 (March 17) 1950.
10. Hall, G. E., and Lucas, C. C.: Cholinesterase activity of normal and pathological human sera, J. Pharmacol. & Exper. Therap. *59:*34 (Jan.) 1937.
11. (a) Hoffman, W. S.: Photoelectric determination of potassium in minute quantities of serum, J. Biol. Chem. *120:*57 (Aug.) 1937.
    (b) Sideris, C. P.: Adaptation of indirect method for potassium to photoelectric colorimeter, Indust. & Engin. Chem. (Anal. ed.) *14:*821, 1942.
12. Whitehorn, J. C.: System of blood analysis, Supplement II. Simplified method for determination of chlorides in blood or plasma, J. Biol. Chem. *45:*449 (Feb.) 1921.
13. Hawk, P. B.; Oser, B. L., and Summerson, W. H.: Practical Physiological Chemistry, ed. 12, Philadelphia, The Blakiston Company, 1947, p. 625.

14. Wolfson, W. Q.; Cohn, C.; Calvary, E., and Ichiba, F.: Studies in serum proteins, V. A rapid procedure for estimation of total protein, true albumin, total globulin, alpha globulin, beta globulin and gamma globulin in 1.01 ml. of serum, Am. J. Clin. Path. *18:*723 (Sept.) 1948.

15. Huggins, C., and Talalay, P.: Sodium phenolphthalein phosphate as a substrate for phosphatase tests, J. Biol. Chem. *159:*399 (July) 1945.

16. Kolmer, J. A., and Boerner, F.: Approved Laboratory Technic, ed. 4, New York, D. Appleton-Century Company, 1945, p. 829.

17. (a) Hanger, F. M.: Serological differentiation of obstructive from hepatogenous jaundice by flocculation of cephalin-cholesterol emulsions, J. Clin. Investigation *18:*261 (May) 1939.
(b) Hanger, F. M., and Patek, A. J.: Cephalin flocculation test in cirrhosis of liver, Am. J. M. Sc. *202:*48 (July) 1941.

18. (a) Maclagan, N. F.: Thymol turbidity test as an indicator of liver dysfunction, Brit. J. Exper. Path. *25:*234 (Dec.) 1944.
(b) Ley, A. B.; Lewis, J. H., and Davidson, C. S.: Quantitative determination of the thymol turbidity reaction of serum, J. Lab. & Clin. Med. *31:*910 (Aug.) 1946.

19. Kolmer, J. A., and Boerner, F.: Approved Laboratory Technic, ed. 4, New York, D. Appleton-Century Company, 1945, p. 831.

20. Koch, F. C., and Hanke, M. E.: Practical Methods in Biochemistry, ed. 4, Baltimore, Williams and Wilkins Company, 1943, p. 318.

21. Sheard, C., and Sanford, A. H.: A photoelectric hemoglobinometer; clinical applications of the principles of photoelectric photometry to the measurement of hemoglobin, J. Lab. & Clin. Med. *14:*558 (March) 1929.

WRINKLE-FREE STOCKINETTE FOR USE UNDER CASTS

WILLIAM B. FISCHER, M.D.
LOUIS W. BRECK, M.D.
El Paso, Tex.

THE use of stockinette as the inside lining of casts has long been universally accepted and used. One of the few objections to its use has been the matter of wrinkling into uncomfortable, and occasionally dangerous folds, or the stretching of the stockinette over prominences, producing pressure points. These complications not infrequently necessitate changing a cast at an inopportune time during the course of immobilization.

The accompanying drawings show a method of covering four different regions of the body with stockinette (fig. 1). In the illustration (A) shows a lining for a thumb spica made from the appropriate width of stockinette ordinarily used to cover the forearm. At the distal end a "V" shaped line of stitching is made, located to one side of the middle. This permits the passage of the thumb on one side and the palm of the hand on the other side of the "V." The web between the thumb and the palm is cut out. Thus, a piece of stockinette is formed which resembles a mitt from which the finger tips have been removed.

The stockinette lining for the shoulder spica (B) is made from a length of stockinette of the proper width for the chest which will be long enough to reach from the hand to the hips. A long gently curved stitching is placed at an appropriate distance from the edge to allow the passage of the arm, and it is ended at the place where it will form the lining about the base of the neck. The upper and outer side portion is then cut free and the stockinette applied as a one-arm sweater. For the lower extremities to be fitted with either a half or full spica (C) the appropriate width of stockinette is selected to cover the trunk, with its length that of the trunk and the leg. Next the outline of either one or both of the legs is stitched, and the remaining portion is removed as illustrated.

Body jackets including the head and neck (D) are lined with stockinette of appropriate size for the chest which has been stitched to resemble a "turtle neck" sweater of which the neck portion has been elongated to fit over the head. The redundant portion lateral and superior to the shoulders is removed. While the cast is being applied and trimmed the patient is made comfortable by a small vertical slit through which his nose projects.

When employing the routine technic of application of a plaster of

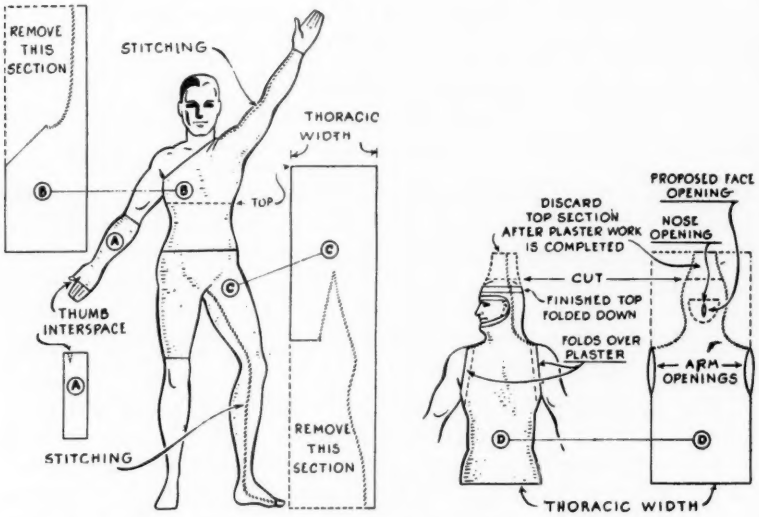

Fig. 1. Drawings illustrating the method of preparing wrinkle-free stockinette for use under casts.

paris cast, with first the stockinette and then the sheet wadding, one encounters no difficulty with the seam formed by the stitching. An adequate stockinette margin can be obtained to cover all exposed plaster edges. This turned back edge is held with the last finishing turns of plaster bandage and incorporated back into the cast, thus making a neatly trimmed edge.

REFERENCES

1. Atkinson, E. W.: Plaster Casts: Their Preparation in the Hospital, Walpole, Mass., Lewis Manufacturing Company, 1937, p. 33.
2. Compere, E. L., and Banks, S. W.: Pictorial Handbook of Fracture Treatment, The Year Book Publishers, 1947, p. 39.
3. Geckeler, E. O.: Plaster of Paris Technic, Baltimore, Williams & Wilkins Company, 1944, p. 11.
4. Key, J. A., and Conwell, H. E.: The Management of Fractures, Dislocations and Sprains, ed. 4, St. Louis, C. V. Mosby Company, 1946, p. 33.
5. Lambert, C. N.: Plaster of Paris technic for application of casts, S. Clin. North America *28*:241 (Feb.) 1948.
6. Luck, J. V.: Plaster of Paris casts: experimental and clinical analysis, J.A.M.A. *124*:23 (Jan. 1) 1944.
7. Quigley, T. B.: Plaster of Paris Technic in the Treatment of Fractures and Other Injuries, New York, The Macmillan Company, 1945, pp. 7, 27.

Drawings illustrating the method of preparing wrinkle-free stockinette for use under casts.

THE AMERICAN SURGEON

Official Publication of
THE SOUTHEASTERN SURGICAL CONGRESS
THE SOUTHWESTERN SURGICAL CONGRESS

Published Monthly by
The AMERICAN SURGEON PUBLISHING COMPANY
701 Hurt Building, ATLANTA 3

Subscription in the United States, $8.00

Volume XVII October, 1951 Number 10

INJURIES TO THE HAND

The frequency with which injuries to the hand are encountered in industry is well known. They are paramount both in their seriousness and the frequency with which they occur. It cannot be too well emphasized that in this day of industrialization the hand of the worker should be considered above everything else. Protection and education have been developed in the prevention of eye injuries, but sufficient education, both to the employer and employee, in the protection of the worker from hand injuries has not been made.

An educational program can diminish further injury and prevent sequelae after the trauma has once been inflicted. Many minor wounds are thought insignificant and treated as such, only to have extensive infections develop, and disability occur as a consequence. Final results are ultimately dependent upon the extent of the injury, the type of emergency treatment instituted, care during the recovery phase, and the reparative or final reconstruction.

Immediate care should be directed toward making a rapid evaluation of the magnitude of the injury, and toward seeing that the proper care is instituted. Many minor wounds can be treated in the dispensary or emergency clinics if strict surgical principles are ob-

served. The attendants must quickly cover the wound with large, protective dressings, which not only prevent infection, but also avoid further injury, allay the pain, and control the bleeding. The problem of hemorrhage has perhaps been given considerable importance. Most instructions in first aid emphasize the necessity of the tourniquet. When bleeding is uncontrollable, the use of the tourniquet *is* demanded, but its abuse is of equal danger. Seldom is it necessary to use in hand injuries, but a large, ample, bulky type of dressing should be applied to the entire hand over a well fitting splint. The immediate use of chemicals for antiseptics is wholeheartedly condemned. These not only do little good, but often add to the disability and permanent injury. Much normal tissue may be destroyed which could otherwise be utilized in the processes of repair.

Time is of much significance in determining the extent of primary repair of the wound. In this age of rapid transportation and close proximity to adequate facilities, this should be no particular problem. Severed tendons should not be sutured if more than four hours have elapsed since initial injury. Nerve sutures are also secondarily done if not seen early. This does not mean that the eventual results may not be good, but it is quite obvious that the earlier the repair, the greater the likelihood of adequate restoration of the injured part exists. Particular emphasis is made on this point to caution us, as surgeons, to consider them as emergency, and to perform the indicated surgery immediately.

There are certain factors which are remembered by the surgeon, and should be emphasized to the patient, the employer, and the insurance carrier. The hand is a very complex structure, an intimate knowledge of which is required if a maximum return to function is to be accomplished. The principles involved are no different from those involved in all major surgery, and it cannot be too strongly emphasized that the immediate treatment of a hand is a major problem. It is well, therefore, that this should be done under the most advantageous circumstances. Considerable time and attention to minute details will be rewarding to all concerned. There are no short cuts in restoration of the damaged hand to its full usefulness in this period when the skilled laborer is at a premium.

The exploration of a wound should not be made except in the operating room. For that reason, a complete diagnosis of the extent of injury is never made until the patient is fully anesthetized.

After a complete evaluation, certain principles are utilized in the process of restitution. A carefully controlled touniquet must be used in order to have a dry field of operation. It must be emphasized that preservation of all viable tissue is most important at this

stage. A thorough debridement must, however, be made if the repair is to proceed without infection. The suturing of tendons and nerves can only be made in a reasonably clean field in which the blood supply has not been impaired. If more than four hours have elapsed, neither of these structures should be repaired since they have been exposed to possible infection too long for safety. For that reason, immediate attention is in order for maximum results. Closure of all open wounds can then be done after thorough debridement of the wound. The use of copious quantities of normal saline is in order, along with the use of mild detergents, but never strong antiseptics. The cleansing may be gentle but complete. This includes the surgical removal of all loose soft tissues which have been devitalized. Only then should the wound be closed. This should be the aim in each instance, even though the tendons and nerves cannot be repaired. Delayed suturing can be successfully accomplished at a more suitable time. The immediate use of a skin graft may be in order when the loss of soft tissue is extensive.

Complete healing of the wound is necessary before undertaking any type of plastic or reconstruction of the hand. Sufficient time must be allowed in order that restoration can be maximum. The reconstruction must of necessity be limited to the extent of injury and the deformity that has occurred as a consequence therefrom. All efforts should be primarily directed towards the functional results.

The hand must be fixed only long enough to allow healing of sutured tendons before undertaking active exercises. When splints are used for too long a period, adhesions and fixations occur, which may be permanent. At the end of three weeks, fibrous union of a tendon for the most part is achieved. Exercises may be allowed only under close supervision to prevent late separation of the repaired parts. During the recovery following nerve suture, the muscles of the hand must be protected and kept in a state of activity to prevent atrophy from disuse, which would in turn result in some permanent deformity.

Physical therapy is in order during this prolonged period of recovery. Constant supervision and direction until satisfactory results are achieved will be rewarded by a more useful and less disabling hand.

J. D. MARTIN, JR., M.D.

Atlanta, Ga.

BOOK REVIEWS

The Editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

AN ATLAS OF ANATOMY. By J. C. BOYLEAU GRANT, M.C., M.B., Ch.B., F.R.C.S. (Edin.), Professor of Anatomy, University of Toronto. Baltimore, Md., Williams and Wilkins Co., ed. 3, 1951. Cloth, $12.00. 637 diagrams.

This is one of the finest atlases we have seen. It covers the entire field of gross anatomy. The illustrations are easily readable and the legends are quite adequate as it appears in its third edition.

These pictures are made in the following manner: each specimen was photographed and then an enlargement was made; with a view box the outlines were traced; these tracings were compared with the original specimens and then given to the artist. The artist worked up the final drawings with the original dissection beside her. With this procedure practically no liberty was taken with the anatomy except the unimportant, confusing minor details have been omitted, giving emphasis to the more important structures.

The diagrams are in illustrative color which, of course, is not necessarily the true color: *i.e.*, nerves are yellow, while the urinary and biliary tracts are green; the arteries red and the veins are blue. Nomenclature is in the Birmingham revision (B.R.) of the Basle Nomina Anatomica (B.N.A.).

This edition contains more than 70 illustrations which are entirely new and 28 of the old illustrations which have been improved are replaced. It comprises a beautiful volume which should be quite worthwhile to the student whether he be undergraduate or post graduate.

A. H. LETTON, M.D.

INTRODUCTION TO SURGERY. By VIRGINIA KNEELAND FRANTZ, M.D., Associate Professor of Surgery, College of Physicians and Surgeons, Columbia University; Associate Attending Surgical Pathologist, Presbyterian Hospital, New York, and by Harold Dortic Harvey, M.D., Assistant Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University; Associate Attending Surgeon, Presbyterian Hospital, New York. Oxford University Press of New York. 1951. 233 pages.

This small book which may be termed a monograph has been written for the medical student to introduce him to the subject of surgery. It might be known as a primer of surgery.

In this small volume the A B C's, the basic principles of surgery, are defined and discussed. The majority of the volume deals with inflammation and wounds and their repair. There are several pages toward the end which deal with the contact with the patient as well as other things, such as anesthesia, care of the patient and plastic surgery.

The volume is rather small. The paper is rough. The printing is fairly small also, but it is easily readable. The style is clear and concise, and the student should find a great deal of information which will help him understand the complex problems of surgery. The volume might also be used in teaching surgery to nurses.

A. H. LETTON, M.D.

Books received are acknowledged in this section, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

THE MANAGEMENT OF FRACTURES, DISLOCATIONS, AND SPRAINS. By John Albert Key, B.S., M.D., St. Louis, Mo., Clinical Professor of Orthopedic Surgery, Washington University School of Medicine; Associate Surgeon, Barnes, Children's, and Jewish Hospitals and H. Earle Conwell, M.D., F.A.C.S., Birmingham, Alabama, Associate Professor of Orthopaedic Surgery, University of Alabama School of Medicine; Chief of the Orthopaedic Service, South Highland Infirmary; Consulting Orthopaedic Surgeon to Carraway Methodist Hospital and Baptist Hospitals; Attending Orthopaedic Surgeon, Children's Hospital, Jefferson-Hillman Hospital, East End Memorial Hospital, and St. Vincent's Hospital, Birmingham, Alabama, ed. 5, St. Louis, The C. V. Mosby Company. 1951. $16.00.

ANNUAL ASSEMBLY OF THE SOUTHEASTERN SURGICAL CONGRESS

The next annual assembly of The Southeastern Surgical Congress will be held in Atlanta, Georgia, at the Biltmore Hotel, March 10, 11, 12, 13, 1952, jointly with The Atlanta Graduate Medical Assembly.

The surgical lectures will be held in the Georgian Ballroom of the hotel and the medical lectures in the exhibition hall, and as far as possible the lectures will run simultaneously.

Please make your reservations early.

ABSTRACTS FROM CURRENT LITERATURE

THE COLOSTOMY: ITS RECONSTRUCTION AND CARE. Frederick B. Campbell and William C. Schaerrer. *Journal of the American Medical Association 146*:93-96 (May 12) 1951.

The authors, favoring a colostomy stoma flush with the abdominal wall describe a method of reconstruction for effecting such an opening. Its advantages are less mucosal trauma, a drier stoma, simplified dressing, minimal stenosis and better patient acceptance. The description of the technic employed is quoted:

"The type of anesthesia is optional, but the usual procedure is the local infiltration of 1 per cent procaine hydrochloride solution. An incision is made through the skin, but as near the bowel as possible. The size of the opening in the skin may be determined by the distance of the skin incision from the bowel. If a tendency to contraction is shown, a larger skin opening should be made. After the skin has been incised, the protruding portion of bowel is then amputated at that level. Two or three vessels will require a fine tie. A short cuff or mucosa is left to afford easy apposition to the skin without undue tension. Mucosa is then sutured to the skin with a lock stitch, using 0 surgical gut on a sharp cutting needle.

"More accurate apposition is obtained by passing the needle in through the mucosa and out through skin. This step should be done with meticulous care because it is the accurate apposition of mucosa to skin that minimizes scar formation and thereby minimizes contraction. The cautery is not used for amputation because of the resulting inflammatory reaction and the tendency to stenosis.

"Mucosa accurately sutured to the skin with a minimum of trauma and no area left to granulate results in very little scar tissue or contraction. Traumatized tissue, inaccurate apposition and a granulating surface may result in stenosis whether mucosa protrudes above the skin or recedes below it."

A simplified plan of colostomy management is described in which simple and readily obtainable apparatus is employed. Plastic refrigerator bowl covers are utilized instead of colostomy domes and are held in place with a wide elastic band supporter. The method of regular irrigation combined with constipating diet is advocated. The authors emphasize that the surgeon's job is not finished with the creation of a colostomy but that it is his duty to adequately train the patient in colostomy care.

R. H. S.

PROGNOSIS OF GASTRIC CARCINOMA; EFFECT OF EXTENT OF RESECTION. Walton D. Thomas, John M. Waugh, and Malcolm B. Dockerty. *Archives of Surgery 62*:847-855 (June) 1951.

A mere philosophy of radicalism is not sufficient grounds for designation of total gastrectomy as the treatment of choice in all carcinomas of the stomach. The worth of the procedure should be critically evaluated from every possible angle before it is generally adopted. In the one reported series (Scott and Longmire) wherein total gastrectomy was carried out for large and small cancers alike over a period of several years, the results were disappointing.

"A properly performed subtotal gastrectomy should remove the entire lesser curvature, including the gastrohepatic omentum. The left gastric artery

should be ligated at the celiac axis, and the entire left gastric artery along with the coronary falx in which it runs forward to the stomach, should be removed. The short gastric vessels should be ligated as close to the spleen as possible. The entire gastrocolic ligament should be removed. Two and one-half cm. of duodenum should be removed where possible, particularly when the tumor abuts on the pylorus. The infrapyloric nodes must be removed completely in all cases regardless of the location of the tumor or the apparent involvement of these nodes. The spleen and the omentum below the colon may be removed if one feels that this is desirable."

No operation for cancer of the stomach is a radical one in the sense that all the tributary lymph channels and nodes, even of the primary type, are removed. Once the tumor has begun to invade lymph channels, successful radical extirpation is achieved only by chance.

In endeavoring to determine if these facts were borne out pathologically, the authors have selected a group of cases with lesions of a sufficient degree of similarity to warrant evaluation of the method of treatment. Of all the cases of gastric cancer removed at Mayo Clinic from 1920 to 1940, 147 cases where selected with lesions of similar location in the prepyloric region, of similar size and degree of pathological malignancy. The gross specimens were carefully measured and divided into groups based on narrow and wide resections. The margin selected for division of the two groups was arbitrarily set and 3 cm. above and 2 cm. below the lesion. Several methods were used in determining the extent of the resection and all yielded similar results. The conclusions drawn from this study are quoted in part:

"Including all cases, the prognosis in those in which resection was done 3 or more cm. above the tumor and 2 or more cm. below was not statistically better than those with more limited resections. When only those cases with involved nodes were considered, the patients in whom wide resections were done had approximately two and one-half times as many five year cures as those in whom narrow resections were performed. Whether or not nodal involvement is suspected, a careful subtotal gastrectomy should be performed in all cases with thorough removal of every known area of lymphatic spread. The pathologic characteristics do not justify total gastrectomy in lesions of the type studied herein. It is conceivable that total gastrectomy might be justified for even small cancers of the scirrhous or invasive types. An improperly performed total gastrectomy might actually remove less lymph node-bearing tissue than a properly performed subtotal gastrectomy."

R. H. S.

SCALENOUS ANTICUS SYNDROME; UNUSUAL DIAGNOSTIC AND THERAPEUTIC ASPECTS. William D. Holden, John A. Murphy and A. Frank Portmann. *American Journal of Surgery 81*:411-416 (Apr.) 1951.

Forty-two patients presenting 45 scalenus anticus syndromes have been seen by the authors and form the basis for this study. A breakdown of signs and symptoms reveals approximately the same percentages of occurrence of neurologic and vascular signs as those previously reported by various observers. Only 9 patients had a cervical rib or sufficient elongation of the transverse process of C7 to consider this factor in the pathogenesis. Muscular exertion stands out as a causative factor. Traumatic myositis of the muscles of the shoulder girdle must be differentiated from the superior type of scalenus anticus syndrome.

The authors emphasize the differentiation of the acute and chronic types of the syndrome. The acute type is usually related to trauma and most frequently responds to rest, heat and procaine injections. The chronic type most frequently results from prolonged and repeated muscular exertion and stretching involving the muscles of the neck and shoulder girdle. These usually require scalenotomy.

In this series, 28 patients were subjected to scalenotomy, or some operative procedure such as resection of the cervical rib. Some patients had anterior thoracic sympathectomy in conjunction with the scalenotomy. Five patients have been advised to have scalenotomy and 9 have responded to conservative measures. Two patients were re-explored because of recurrence of the pain.

In employing procaine injection as a diagnostic test, it is important that the stellate ganglion not be anesthetized until injection of the muscle has been evaluated. If injection of the muscle fails to produce relief and relief follows subsequent injection of the stellate ganglion, anterior thoracic sympathectomy is indicated.

Three complications were encountered. The pleura was opened in one patient followed by mild subcutaneous emphysema lasting a few days. A lymph vessel (apparently not the thoracic duct) was damaged in one instance and required ligation. The third complication occurred in a patient being explored for traumatic brachial plexus neuritis and is included here because of the similarity of the approach. Upon incising the platysma in this patient, profuse arterial bleeding occurred. This proved to be due to avulsion of the thyrocervical trunk from a thin, sclerotic subclavian artery apparently due to hypertension and rotation of the head.

Two patients had postoperative recurrence of symptoms and re-exploration failed to relieve them.

R. H. S.

ANNOUNCEMENT OF VAN METER PRIZE AWARD

The American Goiter Association again offers the Van Meter Prize Award of Three Hundred Dollars and two honorable mentions for the best essays submitted concerning original work on problems related to the thyroid gland. The Award will be made at the annual meeting of the Association which will be held in Saint Louis, Missouri, May 1, 2 and 3, 1952, providing essays of sufficient merit are presented in competition.

The competing essays may cover either clinical or research investigations; should not exceed three thousand words in length; must be presented in English; and a typewritten double spaced copy in duplicate sent to the Corresponding Secretary, Dr. George C. Shivers, 100 East Saint Vrain Street, Colorado Springs, Colorado, not later than March 1, 1952. The committee, who will review the manuscripts, is composed of men well qualified to judge the merits of the competing essays.

A place will be reserved on the program of the annual meeting for the presentation of the Prize Award Essay by the author, if it is possible for him to attend. The essay will be published in the annual Proceedings of the Association.